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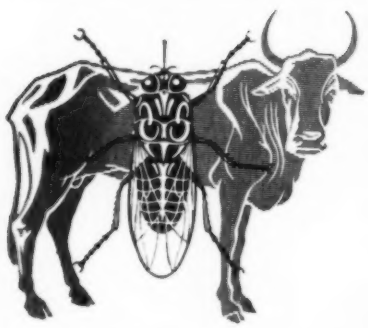

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The President's New Year Message

IT is my privilege, on this day of the year, to wish to every member of the British Veterinary Association, and all other readers of THE VETERINARY RECORD, a very happy and prosperous New Year; and on behalf of Mrs. Beynon and myself I do so most cordially.

New avenues are opening before us, our profession has the opportunity both of serving the community more fully, and of increasing its own knowledge and effectiveness in the light of discoveries made by our research colleagues, veterinary medical and pharmaceutical, in their varying fields of activity. Because of the present need for economy, the swine fever eradication scheme will start a little later than had been hoped, but 1962 will see the inauguration of the scheme for free vaccination against brucellosis, thus opening to us an enlarged field of service to the farmer, and at the same time contributing to public health by reducing the risks of undulant fever in man.

As yet we do not know how the Common Market negotiations will go; but members of the profession may rest assured that the B.V.A., and, no doubt, the Royal College, will follow with close attention all developments which may have veterinary repercussions.

In any case the possibility of still closer relationships with our continental colleagues will be grateful and stimulating both personally and professionally, and now THE VETERINARY RECORD has become an international journal I should like also to extend my good wishes to our many friends in Europe.

The New Year is, by tradition, a time for fresh resolutions; but there can be no harm in reflecting, with thankfulness, upon the assets we already enjoy. At the turn of the year our membership is the highest ever recorded; new bodies having specialised interest —small-animal, equine, zoological and so on—have come, or are coming into existence, thereby attesting the keen pursuit of ever-widening knowledge; THE VETERINARY RECORD is more widely circulated than ever in its history, and I have learned, with the greatest pleasure, that our new journal, *Research in Veterinary Science*, is on sale in no fewer than 72 countries.

Besides greeting members of our own profession both here and overseas, I should like to send my good wishes to our many friends in scientific and agricultural organisations, in trade associations, and in commerce. In the latter connexion I cannot fail to draw attention to the great amount of experimental and research work done by the pharmaceutical houses, whose products are indispensable to the day-to-day work of veterinary surgeons.

Acknowledgment of service rendered leads one to recognise with gratitude all that is done voluntarily by scores, if not hundreds, of busy people for our own Association. My fellow Officers, members of Council and of standing committees, contributors to our journals and handbooks, and a host of others have laid the B.V.A. deeply in their debt, and I feel sure that the general body of members would wish me to express very sincere thanks on their behalf.

Let us, then, leave the old year with a sense of gratitude for things achieved and for future projects advanced, and go forward with hope, gladness and confidence towards what lies ahead.

Symposium on Poultry Diseases

1.—The Clinical Diagnosis of Poultry Diseases

BY

W. P. BLOUNT
British Oil & Cake Mills Ltd.

Preface

IT is no exaggeration to say that one could easily write a book about the diagnosis of poultry diseases, and therefore the difficulty of compressing the subject into one session lasting less than an hour will be appreciated.

In consequence I have decided to pay special attention to the signs and symptoms of disease, neglecting almost entirely the associated subjects of flock histories, *post-mortem* findings and laboratory procedures. This somewhat limited field has been chosen primarily because the veterinary surgeon in practice must know more about it if he is to play an increasingly important part in diagnosing disease on the farm.

I hope to show a series of slides and recordings to make the subject attractive, educational and interesting, but it should in no way be thought that the subject has been dealt with exhaustively.

Introduction

As is the case with other species, the clinical diagnosis of disease in poultry is dependent upon a knowledge of their symptoms and *post-mortem* findings preferably in association with an adequate case history. Armed with this information the veterinary practitioner can make a fairly accurate diagnosis on most occasions, although he is, of course, naturally dependent upon laboratory facilities for confirmatory bacteriological or other examinations.

One of the differences between poultry and other farm animals is the fact that they often exist to-day in flocks of up to 40,000 birds, and in consequence it is commonplace to find half a dozen different pathological conditions affecting any small group of birds sent in for *post-mortem* examination. Furthermore, since the recent move by many progressive poultry farmers towards intensive systems of poultry husbandry, and the necessity from economic reasons for allowing each bird only a small amount of floor space, the rapid spread of certain diseases has been greatly facilitated.

Symptoms (signs) alone are sometimes highly significant, as is true of many cases of neuro-lymphomatosis (fowl paralysis), but the value of a *post-mortem* examination not only allows the affected nerves to be seen, it also gives the veterinarian the opportunity of seeing what other conditions are complicating the disease picture, *i.e.* intestinal worms, coccidia, etc.

Whilst a flock history may suggest that a particular malady is present, *e.g.* pullorum disease, quite often no confirmatory *post-mortem* findings may be obtained until a large number of dead chicks have been examined. The student may wonder, in fact, why this procedure is necessary, since a bacteriological examination will have to be carried out in any case in order to recover the causal organisms. But the answer rests on the fact that if definite *post-mortem* clues can be obtained, such as the characteristically enlarged liver, lung nodules, solid caecal cores, haemorrhagic inspissated yolk sac, etc., the farmer can then be advised to dose his flock immediately with furazolidone (0.04 per cent.) for 10 days without waiting 48 hours or so for the receipt of a laboratory report. There is no question that whenever this drug is being used for curative purposes the earlier the treatment can be given the better.

All this means that whenever possible the practitioner should examine an affected group of birds rather than individuals, particularly since, as pointed out earlier, a single bird is seldom representative of a flock as a whole. Incidentally, the wide variety of diseased organs exhibited by poultry will provide an interesting field to veterinary surgeons who have not been in the habit of carrying out poultry *post mortems* on any big scale.

A second point to emphasis is the fact that specific diseases do not account for more than 30 per cent. of all deaths; the remainder being non-specific, many being related to various aspects of husbandry. For example, in young chicks and poults far more will be found to be suffering from chilling or digestive disorders than from pullorum disease, aspergillosis, etc.

The incidence of frank disease in a flock will be related in part to the extent to which farmers practice culling, and note must always be taken of this fact. The astute poultryman never likes to see any of his birds off-colour so he promptly culls them, and as a result when the flock is visited by a veterinary surgeon they may all look quite healthy. All too often the culls will have been disposed of without being subjected to any investigation, and in this way valuable *post-mortem* material will have been lost, for such birds often give quite a clear indication as to the extent to which the flock is affected by worms, intestinal coccidiosis, gizzard impactions, enteritis, etc.

There are 2 main aspects to poultry disease diagnosis (1) that associated with flock visits where live birds can be inspected, and where the poultryman can usually give a reasonably accurate account of the case history; and (2) that which more commonly occurs following the receipt of a limited number of dead birds for *post-mortem* examination—as often as not unaccompanied by any lucid account of the trouble. Whilst the examination of live birds is valuable enough, the writer generally places greater reliance upon *post-mortem* examinations.

Papers presented to the Annual Congress of the British Veterinary Association in Oxford on Friday, September 8th, 1961.

When practitioners who are unfamiliar with poultry *post-mortem* work open up birds they sometimes fail to gain any satisfaction from them, possibly because they do not have sufficient experience to interpret the picture presented. The state of the bird's feathering, vent, comb, beak and nails, crop, gizzard and the condition of the intestines can all give valuable clues as to management; and, of course, mismanagement often accounts for far greater financial loss than does disease. This does not mean to say, however, that even an experienced pathologist will not have 10 per cent. of undiagnosed cases, but should this figure rise to 20 per cent. or more something is usually being missed.

Aids To Diagnosis

A keen eye, ear and nose. An enquiring mind. Good powers of perception.

Auscultation

Auscultation is of no value for checking the heart beats in poultry as these are too rapid for diagnostic purposes. (Male chicken 240+; duck 200+; turkey 90+ beats per minute.) But auscultation of the upper abdomen will detect clearly the rhythmic grinding sounds arising from the movements of the gizzard and contents, especially if grit is present. The ear is as satisfactory as a stethoscope for the detection of these sounds, but if lousiness is a problem the latter is to be preferred.

Instruments and Equipment

Stout dissection scissors, forceps, clinical thermometer, microscope, light meter, smoke candles, etc.

Palpation

As an aid to diagnosis palpation is sometimes valuable. The gizzard and abdominal organs can readily be palpated if the bird is held by the wings. Alternatively, entrance to the rectum and oviduct can be effected *via* the vent, for palpation of the abdominal contents. Palpation of the crop is necessary to determine the state of its contents, whether normal, pendulous, sour or impacted.

Reflexes

The leg reflexes are best determined if the bird is suspended by its wings which should be held in one hand whilst the other taps the front of the feet in turn. The perch reflex can also be demonstrated by pretending to drop the bird on the floor. A loss of such reflexes occurs in most cases of true fowl paralysis, involving the sciatic plexus, but there are, of course, other causes.

Normal and abnormal movements of the wings can be demonstrated by holding the bird's feet in the right hand (supporting the breast with the left) and "throwing" the bird. The pattern of the flutter of the wings will soon show if there is any brachial paralysis, etc.

Signs and Symptoms of Disease

From a strictly veterinary point of view one should refer more to the signs than the symptoms of disease, since the latter are of subjective origin. But what do we know about the feelings of poultry, and exactly what would they say if they could talk!

Poultry Symptoms

The symptoms which poultry themselves presumably experience can be divisible into those related to health and others concerned more specifically with disease. Examples are hunger and pain respectively, but the natural urge to bully or fight (associated with good health) sometimes results in disease from injury and cannibalism.

Blindness and Deafness

These two symptoms are fortunately not common, the former giving rise to signs readily recognisable by the trained poultryman.

Boredom

Boredom is seldom seen in poultry housed on range because they always have plenty to occupy themselves with, mainly in searching for food. But boredom is common amongst intensively housed poultry, a feature which they try to overcome by a variety of means. If, for example, too few feeding troughs have been provided they may pluck feathers or eat litter or play around. The latter again sometimes leads to fighting. In laying batteries some idle birds spend time breaking up their pelleted feeds, or flicking water all over the place, or by egg eating. (This latter sign, however, may, in fact, be a response to pain.)

Fear

Fear is a natural instinct and poultry are, of course, afraid of people, a feature normally expressed by their keen desire to escape from the persons concerned, accompanied by loud squawking if they are caught.

Frustration

Frustration is another common symptom, felt doubtless when poultry cannot get to their feed troughs or drinkers as a result of paralysis, *e.g.* in certain cases of epidemic tremors, fowl paralysis, crazy chick disease, etc.

Frustration is also well seen in cafeteria laying battery cages and also in connexion with the use of other automatic feeders. It presumably occurs too when cockerels are prevented from mating, and it is also seen characteristically in hens which have strayed from their enclosure and which are unable to find their way back again through the gap by which they left the compound.

Hunger

Hunger and thirst are two symptoms of great importance and if either are lost for any reason death will follow. Poultry instinctively make great efforts to satisfy their appetites, which in some ways, however, are inextricably linked with their energy requirements.

In nature a fowl's diet is bulky, hence the need for a crop, but there is very little place for a crop to-day. The semi-continuous intake of mashes,

crumbs or pellets (the ingredients of which will have been ground in the mill) means that such feeds will not be held up in the gizzard, and therefore there is no natural bottle-neck at that site. Complications arise, however, when feed intake is restricted by rationing, as is the case nowadays with broiler replacement pullets from 8 to 20 weeks of age. In an effort to get at the limited amount of feed provided, bullying often occurs leading to fighting and cannibalism: hence the necessity for de-beaking. And unless care is exercised semi-impactions of the crop or gizzard may occur through depraved appetites. Scratching amongst the litter for additional nutrients will also increase the risk of internal parasites, *e.g.* intestinal coccidiosis.

Panic

Panic is a further expression of fear, of mass origin, and well seen at night time should a flock become frightened during darkness. The birds usually rush into a corner and some may get smothered. Evidence of panic may be heard in laying battery houses during the evening or in the early morning when one bird becomes frightened, giving vent to a characteristic alarm response. Other members of the flock quickly take up the same call which continues for several minutes before gradually dying down.

Pain

That pain and discomfort (distress when more marked) are two major symptoms of poultry is clear. When chickens prefer to sit rather than walk or run —as is a feature of rickets, synovitis and several other infections involving the hocks—this is presumably in response to pain.

Dejection may be felt when a bird is suffering pain which continues for some time, *e.g.* from frostbite.

Uneasiness is the common external sign related to discomfort, or even pain, observable in many fowls at the time of egg laying (oviposition). The sequence of events, as seen in laying batteries, may be as follows:—The pullet squats, stands up, moves around and obviously fails to get into a comfortable position. It may then actually peck at the wire floors of its cage as a reaction to the abdominal pain felt. It " talks," sits down again. shuffles around uneasily and then adopts a semi-standing or crouching attitude. Straining takes place and a little faeces may be passed, but following further straining an egg will be laid. Immediately afterwards the bird will stand motionless for a while before turning round to start eating and drinking again.

Discomfort presumably also occurs when there is any marked increase in tension within the pericardium or abdomen (from fluid), and from impactions of the gizzard and crop, oviduct, ureters; mild inflammatory conditions like enteritis, early peritonitis, etc. Similarly, there may be irritation from external parasites, but whether internal parasites (worms) also give rise to discomfort is not known.

Segregation

Many sick birds isolate themselves from the flock, a very typical example being the case of a young chick suffering from an infected yolk-sac (" mushy chick " disease). Standing by itself, motionless, dejected and with an obviously swollen abdomen, this attitude is usually diagnostic in affected chicks or poults.

In growing stock many culls segregate themselves, often by retiring to their perches at a time when other members of the flock are busily feeding.

Thirst

Water consumption varies normally according to age, the weather, the composition of the feed and disease. Cold water does not adversely affect water intake in young chicks (Heywang, 1940), whereas warmed water may have this effect. Chicks consume about 2¼ c.c. of water per 1 g. of feed intake; with laying stock the consumption increases 50 per cent., due to water lost in the egg. A White Leghorn consumes about 18 gallons of water per annum, rather more than 7 oz. per day. For heavier laying stock the normal calculation is 5 gallons per 100 birds per day. The total daily water loss for week-old chicks is about 20 g. per bird, and for laying fowls over 400 g. (14¼ oz.) (Medway & Kare, 1959).

In hot weather water consumption increases, for example, at 95° F. water intake is twice that at 70° F.

Feeds which contain above average contents of salt (sodium chloride), potassium or magnesium compounds, or high levels or sulphates, etc., may also lead to increased water consumption. This frequently results in the production of wet droppings. An increased protein content of certain poultry feeds may also influence the intake of water adversely, whereas feeds rich in energy (tallow) may result in drier droppings and lowered intakes of water.

When body temperatures exceed 108° F. water intake increases as is common in all diseases characterised by fever, and as seen also when birds have been poisoned, *e.g.* by phosphorus. Increased thirst may be present in aspergillosis, Keel disease (ducklings), fowl typhoid, blackhead, hexamitiasis, etc.

Constitutional Signs

Bullying

The hierarchy inherent in the so-called " peck " order of poultry (seen more obviously in males than females), means that bullying is a normal feature in any flock which is closely confined. The same poultry on free range would not show much evidence of this natural, dominant, trait.

The introduction of new stock amongst an established flock of birds always initiates a new " peck " order, often to the detriment of the newcomers who frequently get bullied. Bullying is a sign of rude health, but it frequently results in fighting, cannibalism, etc.

It also seems natural for poultry to attack their weaker companions, and it is no uncommon sight to find a sick or dying bird literally being bullied to death from cannibalism.

In adult stock the bird which isolates itself is weak and soon going to die, *e.g.* from TB, lymphomatosis, dropsy or other fatal complaint.

Debility

General debility (asthenia) is an accompaniment of many subacute or chronic illnesses in poultry, particularly those associated with internal parasites. It relates to loss of appetite, impaired digestion and possibly to the absorption of "toxins" produced by intestinal bacteria or by the parasites themselves.

Such stock are generally anaemic, below normal in weight and they very often have a subnormal temperature. If chased they totter about and stumble from sheer physical weakness. When resting their heads sink forwards onto their breasts, as seen in *E. coli* septicaemias, salmonellosis, fowl typhoid (adults), etc.

Fever

At day-old the normal temperature of the baby chick registers only 102° F. gradually rising over the next week or 10 days to 105° F. By the fourth week it averages 107° F. and may even go as high as 108.8° F. by 11 weeks of age. In the laying pullet it is usually about 1½° F. lower, averaging 106.3° F.

The temperature of the day-old (broad-breasted) turkey poult is about ½° F. higher than that of the chicken, rising to 105° F. at 5 days of age and 106° F. by 3 weeks later. After this there is little change, adult turkeys aged 70 weeks having a temperature range from 105.5° to 107.8° F.

As a contrast, 2-day-old ducklings have a body temperature of up to 107° F. averaging 106.3° F. This remains unchanged during the next 2 weeks, but the temperature of ducklings aged 7½ weeks ranges from 106° to 108° F.

In the fowl any temperature over 109° F. can be considered indicative of fever, although it can rise to as high as 112° F. in specific instances, *e.g.* fowl cholera. When the ambient temperature rises to 90° F. there may be a 1° F. rise in the bird's rectal temperature, more particularly when the relative humidity is above 55 per cent. The respiratory rate increases markedly once an outside temperature of 95° F. is reached, whereas the heart rate is but little influenced.

Conversely when metabolism is at a low ebb, as in caecal coccidiosis or chilling, the body temperature falls to as low as 100° F.

Growth Rates

It is important to get some clear indication of the growth rate of the different species involved, since a delayed growth rate is common in many diseases, as well as when there are errors of management (*e.g.* chilling) or nutritional deficiencies (vitamins A, the B complex, lysine, etc.).

Loss of weight, however, may be physiological, *e.g.* in turkeys during the breeding season; or due to diseases such as intestinal coccidiosis, "cancer," nephritis, tuberculosis, hexamitiasis, etc.

Huddling is indicative of hypothermia. Room temperatures may be too low, or disease may be present causing a lowering of the metabolism, *e.g.* chilling, Salmonellosis, etc.

Listlessness

This may be a symptom as well as a sign of ill-health, and can be seen in most illnesses which are characterised by debility, such as salmonella infections, toxic fat disease, mushy chick disease. Very often the birds stand motionless for long periods at a time.

Sleep

Poultry undoubtedly get sleepy from natural causes, but sleep is also a characteristic of certain diseases. The state of relative unconsciousness (with inactivity of the voluntary muscles), is often noted in poultry, but usually only for short periods at a time. Chicks may be found sleeping under their hovers and pullets can also been seen sleeping in their hen battery cages, even during the daytime. Periods of prolonged, sound sleep, however, are uncommon.

An inclination to doze is seen in various diseases, such as B.W.D., Mushy Chick Disease, *E. coli* septicaemia, Fowl Typhoid; "X" Disease, Blackhead and Coccidiosis in turkeys and Anapestifer infection in ducklings, etc.

Sigas Involving the Skin and Feathers

The Skin

Advanced lesions of the skin are not common in poultry, although if there are deficiencies in the diet of the water soluble B vitamins, especially in turkeys (because of their greater requirements), encrustations of the mouth, the corners of the eyelids and feet may result. A dermatitis of the feet from chemical irritation occurs if disinfectants or creosote are used indiscriminately in brooder houses.

Eczematous skin lesions in poultry are rare, but we have recently encountered a number of "burns" at the front of the neck in growing turkeys, without being able to ascertain their cause. Direct spraying under the wings with aerosols containing pyrethrum (for the control of external parasites) or applications of 40 per cent. nicotine sulphate may cause a local exfoliative dermatitis.

Fluid under the skin is seen in exudative diathesis of vitamin E origin, toxic fat disease and salt poisoning.

Haemorrhages under the skin are seen in haemorrhagic disease, also in cases of vitamin K deficiency, smothering and following local injuries.

Bleeding cysts of the skin are commonplace, particularly in "hybrid" pullets, White Leghorns and Light Sussex, and these may readily be mistaken for wounds and injuries. Similar haemangiomata may also affect the liver leading to severe internal haemorrhage, the latter being visible (even before the carcase is opened) at the sides of the abdominal cavity.

Bruising is well seen when poultry have suffered local damage to the sides of the chest and abdomen, etc. This is also a characteristic of many birds which have been smothered and trodden upon by their companions before death took place. (The part is generally denuded of feathers and exfoliation is also common.)

The skin over the pectoral muscles contains many small veins, but these are only obvious when they are congested, as is a feature in many cases of nephritis or visceral gout.

Emphysema is a sequelae to surgical caponisation on occasions, but a non-surgical, genetic form is seen rarely.

When the skin is dry, scurfy and closely adherent to the sternum, it is a sign of dehydration, a feature however which is often better recognised in the legs and feet. This is usually of pathological origin (*e.g.* hexamitiasis, nephritis), but in the case of young chicks and turkey poults one must always be certain of the supply of drinking water, because water starvation is not unknown on badly managed intensive units.

The Breast

The skin covering the keel is liable to local injury following the constant irritation which occurs when adult fowls fail to keep their breast bones raised off the floor or perch. This results in breast blisters, many of which become infested cysts. A cause for breast blisters in broilers, however, is the specific disease infectious synovitis, which must always be carefully differentiated from the more physiological variety of breast blister commonly seen in male chickens (New Hampshire crosses) weighing over 4 lb. at 10 weeks of age. These latter may be associated with a paucity of feathers in the breast region, a condition capable of correction by improved breeding. Before breast blisters are usually obvious in chickens suffering from infectious synovitis, swellings of the main joints may have been noted, particularly the feet, hocks, mandible, etc. These may fluctuate, which assists in their differentiation from swellings (swollen hocks) due to staphyloccoccal infections. Also, of course, from other conditions involving the hocks, such as atypical Pullorum infection in chicks, and the well-known enlarged hocks of turkey poults.

Purplish blotchy lesions of the skin over the breast may be seen in erysipelas in turkeys; the snood is also usually swollen or gangrenous.

When examining the sternal region a crookedness of the breastbone is common. Sometimes it is only dented, at others it has lateral displacements. Although occasionally due to nutritional errors in chicks involving improper levels of calcium, phosphorus or vitamin D in particular (Warren, 1937), in layers it often has a genetic basis (linked with calcium metabolism?). Incidentally, the practical poultryman always associates crooked breastbones with perching at too early an age, but whether this has any foundation in fact is doubtful.

The skin under the toes is thickened, coarse, inflamed or cracked, especially well seen in turkey poults when the diet contains suboptimal amounts of various B vitamins.

The Feathers

Tight feathering is a feature of good health, but loose feathering is not necessarily a sign of disease, although in botulism in ducks the feathers pull out very easily. Under intensive conditions of husbandry, especially during infra-red brooding, or when red lighting is used for laying stock, the feathers often appear rough or broken.

In embryos when the down fails to emerge (clubbed down), this is often linked with a riboflavin x genetic interaction.

Feathers on the shoulder matted with mucus are well seen in coryza, but when blood is involved, blood cysts or local injuries are the cause.

In hen laying batteries some pullets' feathers may appear curled and reminiscent of "frizzle feathers," but the stock are usually perfectly healthy and laying well. At one time it was thought that this condition was due to a zinc deficiency, but that is not the case.

Depigmentation of bronze feathers in young turkey poults is characteristic of lysine deficiency, whereas in chickens and fowls (*e.g.* Bl. Leghorn and R.I.R.) loss of pigment may be related to a folic acid deficiency. The latter, however, has no adverse effect on feather pigmentation in poults, but it may cause instead a cervical paralysis.

When laying stock show bare backs, bare (reddened) abdomens and bare necks, according to one authority these may be an indication that the diet is deficient in methionine. A genetic cause is more usual, but interaction with nutrition probable.

The feathers below the vent frequently show clusters of lice eggs, and if the abdomen at this site is searched carefully, lice or mites can often be seen.

Feathers are easily broken off and for this reason pullets in hen batteries frequently show bare necks or crops, a feature which also occurs when the drinkers or feeders (in intensive houses) have wire guards present for separating the stock. This condition must not be confused with the broken feathers and reddened skin resulting from attacks by depluming mites (*Cnemidocoptes*). Ruffled feathers are frequently seen when stock are ill or diseased (*e.g.* from rickets, B.W.D. coccidiosis, etc.), but fluffed out wings are a normal feature in hot weather, the birds adopting this attitude to keep themselves cool. In cold weather loose feathering is normal.

Feather pecking is commonplace amongst intensively housed stock, and in the layman's eyes this necessarily means that the feed is deficient! As a fact it is not usually a sign of either disease or nutritional deficiency (or excess), it is due primarily to boredom. It is a playful method of occupation, initiated by the more inquisitive members of the flock whose natural instincts urge them to peck at anything unusual at hand. For this reason it generally commences amongst poultry which are inadequately feathered, and the attack may take place at a time when young feathers are sprouting. (This in itself may give rise to slight pain or irritation which makes the bird attacked pay attention to itself in the first instance.) Once a feather has been pulled (pecked or picked) and blood drawn, the attack is often intensified. This can usually be prevented by the use of spectacles, de-beaking, the adoption of a reddened lighting system, or the use of mash instead of crumbs or pellets (mash occupies the birds longer, giving them less time to be bored).

Signs Involving the Head and Neck

Head Shaking

This is a phenomenon which appeals to many poultrymen because of its obvious nature, but it may not have any pathological significance. Such movements shift the position of the pecten, and this is considered to be the normal physiological method for helping to keep the fluid in the posterior chamber at its correct temperature. When head shaking is excessive it may have followed an upper respiratory infection (coryza, I.L.T., etc.), but it is a sign which is quickly imitated by other (healthy) members of the flock. It may also be seen in some cases of Infectious Sinusitis in turkeys. Nodding in turkeys may indicate Newcastle Disease.

The Beak

De-beaking is sometimes so badly carried out on some farms many of the birds appear almost deformed, others die from haemorrhage.

Crossed or other deformed beaks are generally considered to be of genetic origin, but it is surprising how many such birds manage to eat and grow normally.

Impactions of the lower beak (mandible) are often associated with fine particles of mash clogging the mucous glands; an associated feature is curled tongue.

Ulceration of the beak, plus sore feet, are said to be present in cases of biotin deficiency in chicks, and in turkey poults the toes may also slough off.

The Nose

Nasal discharges are typical of "colds" or coryza, and if they reach the stinking stage usually represent what is termed "roup." They may appear also in infectious bronchitis, C.R.D., laryngo-tracheitis and erysipelas infections. Many affected birds wipe their beaks on the feathers covering their shoulders, a feature which should readily be detectable. Infections of the nose may also cause sneezing or snuffling, but when "rattling" or "squawking" are heard these usually relate to upper respiratory infections involving the trachea.

The Eyes and Infra-orbital Sinuses

One or both eyes may show clear-cut evidence of blindness from either keratitis, irido-cyclitis or cataract. Avian encephalitis may be involved. When the eyes are deformed or missing in embryos or chicks, incubation faults are commonly the cause. Pin-point pupils are also seen in some cases of fowl paralysis, but must not be mistaken for cat's eyes pupils, which are a genetic defect. A simple depigmentation of the iris is common in 4-month-old pullets, but prior to this age the normal pigmentation process is incomplete. Dilated pupils occur in nicotine poisoning.

Lachrimation occurs in some cases of poisoning, *e.g.* parathion in conjunction with salivation; by itself it is seen in coryza, fowl pox, local injuries, etc. When associated with vitamin A deficiency a whitish cheesy exudate appears beneath the eyelids, xerophthalmia (dryness of the cornea) and multiple pustules at the back of the throat (extending down even to the crop) are also seen in severe cases. The eyelids may be granular, a feature not to be confused with blepharitis now becoming common in turkeys, but of unknown origin. The eyelids may stick together if the diet is deficient in pantothenic acid, scabs are then also prominent on the eyelids, corners of mouth, etc. Similar scabs may be present in fowl pox, and lesser ones in cases of biotin deficiency, the lids tending to stick together. Yellow pus beneath the lids is a feature of Aspergillosis in turkeys.

Kerato-conjunctivitis is now well-known in young broiler chicks housed on old litter emitting strong concentrations of ammonia. The eyelids may be swollen and corneal ulcers present.

The infra-orbital sinuses are typically involved with P.P.L.O. infections in turkeys, sometimes also in pullets; and they are occasionally obvious also in cases of vitamin A deficiency. The swellings seen in infectious sinusitis are often very great.

The Comb

In the laying fowl and healthy cockerel the comb is bright red and well developed, but it becomes paler and smaller following caponisation or when birds go out of lay. These are all normal features of hormonal origin.

Dubbing is sometimes carried out at hatcheries on day-old pullets, experience in the U.S.A. having indicated that this may be an aid to slightly increased egg production. Dubbed poultry do not suffer from the discomfort associated with the presence of large combs, nor do they suffer from frostbite, a feature often seen in cockerels on range in very cold weather. But dubbed poultry are unable to tolerate hot weather as readily as those which possess a comb, and they may, therefore, be at a slight disadvantage in the summer.

The comb may show both specific and non-specific lesions, one of the best examples of the former being the presence of yellowish fowl pox scabs. The diagnosis can be confirmed by finding typical pox lesions in the mouths of other birds of the same flock. (Pustular lesions at the angle to the mouth are very often confused with those of fowl pox, but do not represent an infectious disease, being present only in isolated instances.) The application of suspect fowl pox material to the scarified comb of a susceptible chicken is seldom necessary, but occasionally required for accuracy of diagnosis, especially when turkey pox is under consideration. The early vesicular stage is seldom seen in fowls or turkeys, but a coalescence of several adjacent lesions is typical of pox.

When the comb is very scurfy and especially if the unfeathered portions of the face are also affected with the same type of white, exfoliative crust-like dermatitis favus is to be suspected; but a non-specific condition can give rise to very similar lesions on the face, although this dermatitis, too, may be of fungal origin.

The pale (small) comb and face so often seen in culled growing stock are generally indicative of anaemia, sometimes secondary to intestinal parasites, whereas in older poultry pale combs (of normal size) are suggestive of internal haemorrhage due to rup-

ture of the liver or spleen. In male turkeys between the ages of 10 and 20 weeks a pale head may signify rupture of the aorta. Rupture of the liver in fowls usually follows advanced fatty degeneration, tuberculosis or heat-stroke. The latter used to be common in pullets housed in the upper row of a unit of laying battery cages immediately below a roof light. It is, however, less common to-day partly because of the higher inclusions of choline, vitamins B_{12} and E in hen battery rations.

A yellow comb and skin generally are characteristic of jaundice, and seen typically in cases of erythromyelosis (fowls) and malaria (turkey poults).

A darkening of the comb, face and head parts generally (cyanosis) is seldom indicative of any particular disease, although it is a feature of many cases of blackhead in turkeys, septicaemias generally, infectious laryngo-tracheitis, fowl cholera, and in some cases also of pullet disease. When isolated birds are affected, such cyanosis may have followed choking, or roundheart disease; but in male birds it may be more typical of heart failure.

Gangrene of the comb in male breeding birds on range in the winter is generally diagnostic of frostbite, although local injuries including fighting, may cause confusion. Gangrene is also associated with poisoning by Ergot, paraldehyde and cantharides. In turkeys a gangrenous snood is seen typically in erysipelothrix infection; male birds first being affected.

The Face

A swelling of the face from oedema is seen in infectious coryza, often associated with haemophilus infection, but when the whole head is involved in cockerels, and turning bluey-green, this suggests an infection following chemical caponisation, possibly due to the use of an infected syringe.

A swelling of the infra-orbital sinus in fowls due to exudate accumulations (coryza) or pus, may be observed in some cases of P.P.L.O. infection and even in fowl pox, but a more marked swelling, of course, is seen typically in infectious sinusitis of turkeys. In poults a vitamin A deficiency is also sometimes involved. One-eyed roup is a classical finding in fowls and whitish pus may also be present under the eyelids, including the nictitating membrane in chronic cases. An association with vitamin A is usually present.

The wattles may be swollen severely, due to the presence of solid yellow, adherent pus. (See later.)

It will also be remembered that a swelling of one wattle is diagnostic of tuberculosis following application of the tuberculin test some 36 to 48 hours earlier.

The Ears

The ears are seldom affected by disease, although when a fowl shows evidence of limberneck it is often necessary to examine the cavity of the middle ear for the presence of solid pale yellow pus—an abscess.

The Mouth

Salivation is a feature of poisoning by parathion.

Diphtheritic or pustular lesions in the mouth, glottis and throat appear in cases of fowl pox, chronic infectious laryngo-tracheitis, infectious coryza, avitaminosis-A and also from local injuries. Pox lesions (yellowish) alone are adherent and bleed if they are removed manually; those linked with vitamin A (whitish) are multiple, characteristically involving the pharynx and oesophagus. Involvements of the eyes and nose nearly always accompany those due to coryza, whilst the history of I.L.T. is generally of a positive nature.

In cases of nicotinic acid deficiency the mouth may be inflamed and the tongue appear deeply congested ("black tongue").

Gasping is not to be confused with panting, the latter being a physiological feature seen in hot weather. (Rectal temperature 108° F. plus.) Gasping in young chicks which are kept intensively may be indicative of aspergillosis, whereas with stock housed on range (including turkey and pheasant poults), one's thoughts turn to gapes. Not, however, if the birds are over 8 weeks of age, because an age immunity will have occurred by that time.

The gasping which may be seen in isolated older pullets may represent fowl paralysis, involving the vagus nerve, whereas if large numbers of birds are affected simultaneously in the classical air-hunger (neck extended) manner infectious laryngo-tracheitis will be suspect. And this is probably the only disease which, in its typical form, is characterised by the coughing up of blood. If an isolated bird is involved and if retching movements are commonplace and especially if the bird squawks periodically, a foreign body in the larynx or trachea should be sought.

Coughing to clear away tracheal mucus is seen in infectious laryngo-tracheitis, infectious bronchitis (chicks) Newcastle disease, infectious coryza, C.R.D., gapes (in association with mouth breathing), etc.

The Tongue and Throat

A marked dryness of the tip of the tongue is termed "pip" and seen when mouth breathing is prominent, as in some cases of roup (Shannon, 1947). Curled tongue has a genetic basis primarily, but may be linked with deficiencies of iso-leucine, leucine or phenylalanine (Grau, 1945). Its occurrence in turkey poults fed mashes can be prevented by the use of crumbs or pellets. Foreign bodies are sometimes found around the base of the tongue (*e.g.* string). Pus lesions are seen at the back of the tongue in vitamin A deficiencies, fowl pox, local injuries, etc.

The Voice

During the daytime the voice of the fowl is heard frequently, being very expressive. It ranges from the well-known soft "cluck-cluck" of the broody hen to the louder, more raucous crow of the cock. Any student of poultry behaviour will soon detect the differences between the voice sounds associated with disturbances, satisfaction, enquiry, pain, etc. The softer sounds are emitted through the nose with the mouth closed, whereas positive movements of the lower beak are always seen to accompany the louder calls. The plaintive high-pitched "cheep" of the constipated chick (typical of B.W.D.) is well-known, as is the somewhat similar note of the chick which

has accidentally isolated itself from its companions.

The voice of the turkey poult is equally impressive, contrasting greatly with that of the chicken.

The Wattles

De-wattling or "cropping" is sometimes carried out in an effort to prevent freezing of the wattles which may occur in winter in cocks of those breeds with long wattles (*e.g.* Leghorns, Ancona, Minorca, Barnevelder, Marans, etc.).

Thickened, deformed wattles (including the space between them) are often due to the presence of solid, yellow pus. This may be secondary to coryza (when pus may also be present in the cleft palate), or related to the chronic form of cholera, in which case Pasteurella organisms are involved. Local injuries are easily recognised. Swollen oedematous wattles occur in acute F. Plague.

The Neck and Crop

Cellulitis of the upper part of the neck is seen in conjunction with local infections, *e.g.* following chemical caponisation. The pellet of stilboestrol may sometimes be felt in this position, unabsorbed, especially in turkeys.

The crop should be examined superficially for the presence of food, impactions with grass, straw, etc. A flabby crop with acid-smelling contents is typical of sour crop. Pendulous crop occurs in turkeys as a genetic defect accentuated by drinking large volumes of water in hot weather. It can be induced experimentally by a diet rich in glucose which aids the growth of various fungi, *e.g. S. tellustris, T. glabrata, C. albicans* (Wheeler, *et al.*, 1957). Some outbreaks of pullet disease are characterised by infections of the crop, in others it is rather pendulous and distended by gases and semi-liquid food contents.

A twisted neck (wry-neck) may be of fowl paralysis origin or related to middle ear disease. A flaccid paralysis of the neck is seen in botulism in ducks, the associated feathers pulling out very easily.

Signs Associated with the Respiratory System

Coughing, sneezing, râles, etc., are obvious in young chicks suffering from a variety of bacterial or virus infections. In conjunction with a profuse nasal exudate and swellings of the facial sinuses they are to be linked with P.P.L.O. (mycoplasma) infections, often followed by an *E. coli* septicaemia after 5 weeks of age. In association with obvious nervous symptoms Newcastle disease is to be suspected, but when the respiratory symptoms are mild and uncomplicated, the mortality low and the spread rapid (the incubation period being very short) infectious bronchitis is probable.

Noisy breathing is a feature best heard at night time when all else is quiet. It is a feature of many outbreaks of Newcastle disease, epidemic tracheitis and infectious bronchitis (chicks). In infectious laryngotracheitis gurgling sounds may be heard or sometimes a high-pitched whistle.

Rapid breathing is characteristic of fevers, or hot weather (80° F. plus).

Gasping in young chicks and poults is seen in Aspergillus infection and gapes; but when isolated birds alone are affected a foreign body may be present in the larynx or trachea. *Air hunger* symptoms in older birds, but with the neck stretched upwards with each inspiration, are typical of infectious laryngo-tracheitis. Bloody mucus may be coughed up in acute outbreaks. If any nervous symptoms are present, however, Newcastle disease should be suspected.

Coughing in turkeys, following swellings of the infra-orbital sinuses, is indicative of an extension of the P.P.L.O. (mycoplasma) infection to the abdomen with air sac involvements.

Laboured breathing occurs when there is increased intra-abdominal pressure, *e.g.* from fluid (dropsy), as seen in toxic fat disease in chickens, etc.

Signs Involving the Legs, Feet and Wings

The Legs and Feet

The colour of the shanks may vary from white through straw to yellow, or even black. All these are normal features linked with the presence or absence of carotenes, xanthophylls or melanin. Similarly, a strong, reddish pigmentation of the hocks is a normal feature in many vigorous cocks (R.I.R. in particular).

The raised scales of the shanks seen typically in scaly leg disease (of *Cnemidocoptes mutans* origin) are well-known, but not to be confused with the coarseness of the shanks in old hens, which are a normal feature.

When both shanks are grossly swollen, but without necessarily causing any marked lameness, this is indicative of Osteopetrosis, seen in older growing or adult stock, and generally to be confirmed by finding similar changes in other long bones, *e.g.* tibia, humerus, etc. In acute cases the parts feel hot and a stilted gait is common. The bones concerned are grossly thickened and the marrow cavity considerably reduced.

A swelling at the base of the shanks in adult birds involving the metacarpal-phalangeal joints is typical of gout, but some cases of bumblefoot may also appear above, as well as below, the foot. In bumblefoot there is generally a site to be seen where the infection has gained access to the subcutaneous tissues. (In broilers any swelling at this site may be a sign of infectious synovitis.)

In embryos and day-old chicks, short, chubby shanks have been linked with a deficiency of manganese.

Any obvious swelling of, or near to, the hocks requires careful examination to distinguish inflammatory lesions (infections from local injuries; pasteurella or staphyloccocal arthritis, chronic erysipelas, synovitis), from rupture of the tendons. This is a comparatively new condition seen in particular strains (both cockerels and pullets) at about 5 months of age. Genetics and nutrition are both involved, but the details of the latter have not yet been ascertained.

Dehydration of the shanks is seen in pullet disease, hexamitiasis, etc., and due to an excessive excretion of water, or alternatively to decreased intake.

The Feet

When the toes have a "clutching hand" effect, due to extensor paralysis, this is often typical of neurolymphomatosis, one foot frequently being involved. When the toes of both feet are doubled underneath in young chicks, so that they walk on their hocks, this suggests vitamin B_2 deficiency, but confusion may arise from crooked toes. When the toes are balled up with adherent litter this is indicative of bad litter management. Long clean toenails are seen in birds which cannot scratch, *e.g.* those housed on wire floors or in laying battery cages; whereas in the case of stock on range the nails always contain adherent earth. (Sometimes a feature of value in legal cases.)

A sloughing of the centre toe in layers housed in hen batteries may follow a local injury to the underside of the toe nail, arising from a metal projection. Several or all the toes may slough away following chemical irritation (disinfectants), a feature also seen in rare cases of biotin deficiency.

Lamenesses and Paralyses

A bowing of legs is common in young ducklings suffering from perosis and it may be associated with "hock" disease in turkeys.

Hock deformities are common in turkeys at different ages, some are genetic, others nutritional or linked with management. In very young poults a temporary enlargement of the hocks may be of nutritional significance, nicotinic acid and vitamin E may be involved; in older poults rickets. In turkeys, aged 10 to 16 weeks, especially heavy, broad-breasted strains on slatted or wire floors, genetic factors usually play a primary part, but many such cases are of ill-determined origin.

A slipping of the Achilles tendon—perosis—may affect only one leg. Manganese, zinc, choline, nicotinic and folic acids, vitamin E and biotin may all play a part in this well-known abnormality in turkey poults, whereas in chickens perosis is usually associated with a manganese deficiency alone. When less than 5 per cent. of stock are involved, however, genetic factors can be considered to be of primary significance.

Whenever a leg or foot joint is involved, lameness is usually a feature, and in some cases paralysis too. The more common causes are:—

Chicks

Curly toe (vitamin B_2), crooked toes (genetic and infra-red ray brooding); rickets (vitamin D_3 calcium, etc.); epidemic tremors (virus); crazy chick disease (nutritional encephalomalacia); dislocations, local injuries, etc. Pullorum disease in turkey chicks may also be characterised by lameness.

Curled toe paralysis may respond to yeast or other sources of vitamin B_2. Crooked toes are deformities which do not respond to nutritional changes; the toes are not curled underneath the feet and there is no mortality. Furthermore, many cases revert to normal after the brooding period has ceased.

In rachitis the mandible is soft and pliable, being capable of simple physical distortion. Affected chicks totter around with a stiff-legged gait, sink to their hocks and show characteristic *post-mortem* changes involving the bones, rib ends, parathyroid glands, etc.

Avian encephalomyelitis (epidemic tremors) and nutritional encephalomalacia (crazy chick disease) are often confused, since they both affect numbers of chicks aged 1 to 5 weeks with similar nervous symptoms and a 15 per cent. mortality. When tremors are present this clearly aids the diagnosis and if the chicks are receiving a palatable, well-formulated diet, the nutritional disorder linked with vitamin E is unlikely to be present. Conversely, if softening and discoloration of the brain are found, encephalomyelitis of virus origin is probably not the cause of the outbreak. A microscopical examination of the parts is necessary in all doubtful cases.

Cervical paralysis (neck thrust forward) is seen in poults suffering from a deficiency of folic acid.

Older Birds

Fowl paralysis, coccidiosis, lymphomatic and other tumours, worms, cysts, synovitis, arthritis, dislocations, fractures, ruptured tendons, local injuries, abscesses and haemorrhages. All these are causes for lameness or paralysis in growing or adult poultry, but in laying stock "cramp" (cage layer fatigue) is another well-known cause.

Without doubt in chickens after the age of 8 weeks one usually suspects the presence of fowl paralysis first, and confirmation can often be obtained by finding enlarged nerves, tumours or other growths causing pressure on part of the nervous system. (This may mean the semi-dissection of the carcase to see the state of the vagus, thoracic, sciatic and other major nerves or their plexuses.) A "clutching hand" effect is often typical of fowl paralysis.

Paralysis of the Wings

A dropped wing is typical of fowl paralysis involving the brachial plexus, but fractures or dislocations are alternative causes. Both wings droop normally in hot weather when environmental temperatures exceed 85° F., but this is only a sign of general debility, as seen in many cases of subacute intestinal coccidiosis, B.W.D. (chicks), fowl typhoid, blackhead, etc.

A fluttering or flapping of the wings is seen as a terminal sign in acute fowl cholera, roundheart disease, etc.

Paralysis of the Tail

A one-sided or dropped tail is uncommon but may be of neuro-lymphomatosis origin, or linked to a deformity of the vertebrae.

Signs Involving the Osseous System

Soft Bones

Poor uneven calcification is seen in young stock suffering from rickets, characterised also by a rubbery condition of the beak.

In older chicks a crooked breastbone is often seen when early perching is encouraged, possibly how-

ever, only in conjunction with the use of rations lacking calcium or vitamin D_3; even so genetic factors are sometimes of primary importance. In layers crooked breasts are not uncommon, but these may be a continuing effect of the same condition seen in growing stock; heavy laying as such does not lead to crooked breastbones, unless faulty management is also implicated.

Shortened bones may be seen in embryos or young chicks when there are dietary shortages of manganese or zinc, but genetic factors are implicated, too, especially when the limbs are also thickened.

Thick bones, especially affecting the shanks (metatarsus) and other long bones of the legs (and wings) are seen typically in osteopetrosis in chickens aged 12 to 20 weeks. The marrow cavity is practically obliterated in many such cases and the bones are extremely hard.

Broken bones result in lameness when the legs are involved, or a drooping of the affected wing when the humerus is affected. Dislocations are rare but also result in lameness: perosis may be a feature.

Ankylosis of the cervical vertebrae is occasionally noted as an insidious complaint in young turkey poults, causing a deformity of the neck.

Enlargement of the hocks is more common in turkeys than chickens, being of either nutritional (vitamin E, nicotinic acid, choline, zinc, manganese) or bacterial (staphylococcal, erysipelothrix) origins.

The Back

A twisted or kinked back may be of genetic (osseous) origin, or a further example of fowl paralysis.

Signs Involving the Abdomen and Vent

Abdominal Distension (Dropsy, etc.)

An accumulation of fluid in the abdomen is rare in young birds, except in certain cases of salt poisoning, and more recently in the newly diagnosed "toxic fat disease" of broilers, in which hydropericardium is also pronounced. In adult stock dropsy is more commonly seen in hens than pullets, the excess fluid visibly distending the abdomen giving it a "dropped" appearance in the live bird. Multiple bowel tumours, or advanced liver or heart lesions usually account for most cases of dropsy.

The adoption of a penguin-like attitude is to be seen in diseases involving the reproductive system characterised by internal laying or chronic peritonitis —dropsy may or may not be present.

In many cases of egg peritonitis the abdomen is full and firm, contrasting sharply with the softer, pliable abdomen of the pullet in full lay. (Such birds show a large, moist vent, a feature also seen in chemically caponised cockerels.)

The vent when attacked by so-called "vent gleet" has a very characteristic, obnoxious odour. The junction between the skin and the lining mucous membrane of the cloaca becomes ulcerated, and pale yellow pus will be found adherent to the parts concerned. There is little or no evidence that vent gleet is either infectious or venereal.

Prolapse and Cannibalism

It is characteristic of the laying fowl that it strains (to increase intra-abdominal pressure) in order to pass its eggs to the exterior, but straining may also occur whenever there is any local irritation. This may be of either intestinal or oviductal origin, and that is why it is so important for the practitioner to discover which of these two organs is at fault. Straining may follow cloacitis (sometimes due to the presence of foreign bodies like insoluble grit), or it may be secondary to an inflammation of the oviduct related perhaps to the retention of the shell membranes. Other causes are abdominal tumours, cysts of the B. of Fabricius, local abscesses, etc. Once pressure or irritation occurs, no matter from what cause, the bird strains and eventually the lower bowel, or the lower oviduct together with the cloaca, becomes everted, resulting in the characteristic clinical picture of prolapse. In many cases the parts are then attacked by other fowls—hence the condition of cannibalism. (*Post-mortem* examination may show that the small intestines have been bitten off within a few inches of their attachment to the gizzard.) Such birds die from a combination of shock and either external or internal haemorrhage.

Diarrhoea

Loose droppings may be related to intestinal coccidiosis, worms, "indigestion," pullet disease, tuberculosis, etc. But wet droppings are also seen in the absence of disease, and in laying stock their level of egg production usually remains high. Diet may be involved (excess of sodium, potassium, magnesium, sulphates, etc.) but even when given the same ration some pullets are affected but not others.

From the farmer's point of view, diarrhoea and "scouring" are synonymous, the presence of the stained vent feathers sometimes being of diagnostic value. When purplish these may indicate that phenothiazine has previously been administered, whereas bloody droppings usually indicate caecal coccidiosis in chickens, although Haemorrhagic Disease is another cause, and isolated cases may be related to an invagination of the intestines.

A yellow discoloration of the droppings is seen whenever there is a major break-down of the red blood cells, *e.g.* blackhead in turkeys. Yellow-green scouring occurs when the liver is involved, such as in big liver disease, fowl typhoid, and also with septicaemias like fowl cholera, erysipelas, Newcastle disease, etc. White staining due to urates is a feature of nephritis, pullet disease and visceral gout; also B.W.D. in chickens.

Green droppings are due to the passage of undiluted bile, and this is a feature whenever starvation is present (in some cases of big liver disease) and in fowl pest phosphorus poisoning, etc. When the feathers are adherent and wet this may be unrelated to disease, and merely evidence of the increased passage of fluid following a hypertonic condition of the intestinal contents.

When the faecal matter is frothy and pale brown, this is a normal feature related to the periodic passage of the caecal contents; but frothy, watery, loose drop-

pings of varying colour are also a sign of intestinal infections, and in the young turkey poult they may also indicate hexamitiasis.

Constipation

A pasting up of the vent with white, faecal material is commonly seen in young chicks aged less than 7 days. Whilst this is sometimes suggestive of pullorum disease or other Salmonella infection, other non-infectious causes are well known, *e.g.* the response by some chicks to rations containing high levels of soya-bean meal. Pasted vents may follow faults in incubation; the condition appearing shortly after hatching.

Occasionally the droppings have a very fetid smell. a feature clearly noted in the mass of faecal matter passed once daily by broody hens, at the time when they are released from their coops.

Signs Involving Egg Production

Enquiries concerning the level of egg production and the quality of the eggs produced is often of great diagnostic value. Daily fluctuations are normal and the general trend is for egg production to fall gradually as the laying year progresses.

Falls in Production

Any sudden fall lasting several days is significant, some being of considerable diagnostic value. The most common causes are infectious bronchitis, Newcastle disease, pullet disease, epidemic tremors and C.R.D. to a lesser extent. Sudden, marked falls in environmental temperatures reaching below freezing point; light failures and water starvation may all result in falls in egg production. Nutritional causes are less common.

Each possibility must be investigated. In infectious bronchitis no nervous symptoms will be present; in Newcastle disease the drop in production (even to zero) is quite dramatic and sometimes this is unaccompanied by any other typical symptoms. In both diseases a crop of soft-shelled or shell-less eggs may occur. But these may also be seen following the administration of sulphanilamide, which interferes with the zinc containing carbonic anhydrase ferment present in the uterus and which is necessary for normal egg shelling purposes.

The fall in production linked with epidemic tremors is usually sharper and shorter than that seen in many other specific diseases and it is only whilst production is falling that the causal virus is excreted into hatching eggs; as a result of which avian encephalomyelitis occurs in the resultant chicks.

When pullet disease is implicated, there may be a response to broad-spectrum antibiotics at high level (100 g. per ton), but several weeks may elapse before production is back to normal again. Any dead birds may show nephritis or visceral gout, impacted crops, liver lesions, etc.

A fall in production from 38 to 50 per cent. has been recorded following the use of rations containing too little phosphorus.

Deformed Eggs

When isolated pullets lay eggs which are deformed these may be related to the presence of fibro-leiomyomata involving the ligament of the oviduct. Alternatively, when a pullet lays 2 eggs within 24 hours one is often deformed and the other weak or soft shelled, both having been present in the uterus at the same time. In any normal flock about 1 per cent. of eggs are laid with soft shells, but the pullets concerned soon revert to normal laying. When many deformed eggs are laid Newcastle disease or infectious bronchitis are suspect, the latter also accounting for rough shelled or ridged eggs.

Cracked Eggs

About 2 to 5 per cent. of cracked eggs is also normal, but when the level increases to 7 to 10 per cent. or more an investigation is needed. Possible causes are: shortage or excess of calcium or phosphorus; a deficiency of vitamin D_3, zinc, manganese or magnesium. In hot weather (85° F.) blood calcium levels and feed intakes are lowered, resulting in weaker shells. Certain makes of hen batteries, especially cafeterias, give rise to many more cracked eggs than normal, but adjustments to the cage floors may sometimes solve the problem. As the laying season progresses some "hybrids" tend to produce weak shelled eggs, but this is a genetic weakness and not easily overcome.

Depigmented Eggs

When crops of eggs appear with white instead of brown or tinted shells, this is usually due to the feed having become contaminated with nicarbazin (0.003 per cent.) about 5 days earlier. At the same time the egg yolks will appear mottled. Normal pigmentation is resumed within one week of the removal of the nicarbazin. The hatchability of eggs resulting from this type of feed contamination is greatly reduced. Alternative causes for depigmented (bleached) eggs are Newcastle disease of infectious bronchitis.

Blood Splashed Eggs

When eggs are laid with spots or splashes of blood, this follows a slight haemorrhage at the vent. It may be seen in particular strains of hybrids. Egg size may be a factor involved: the eggs being too large. Smaller brown markings may indicate the presence of red mites.

Double-yolked Eggs

These can be considered to be a normal feature in many strains of laying pullets. They are evidence of over-activity of the ovary, primarily of genetic (hormonal) origin, often accentuated or triggered off by light. Feed is seldom involved, though excess protein is often considered by the layman to be a cause.

Signs Involving the Nervous System

As these are generally quite spectacular they are readily noticed by farmers and veterinarians alike. They may be characterised by tremors, twitchings, rigors, torticollis, walking backwards, general incoordination, ataxia, staggering, muscle spasm, paralysis, vertigo, etc.

SIGNS OF DISEASE IN POULTRY

RECOGNITION CHART

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
The Abdomen		
—diarrhoea	Non-specific	Many varied causes
—enlarged and firm	Egg peritonitis	
	internal laying: Murky Chick Disease	
—pasted up vent	Constipation, B.W.D.	
—protrusion of viscera	Prolapse, cloacitis	Cannibalism frequently present
—scouring	Diarrhoea	Non-specific
—swollen and flabby, penguin posture	Dropsy	A variety of causes
—ulceration of vent	Vent gleet	Only seen in laying stock
The Back		
—kinked	Genetic or F. paralysis	Rare
Beak		
—crossed	Genetic defect of chickens	Noticed soon after hatching
—impacted	Mandibular disease	Often preceded by infection of associated mucus glands
	"Shovel Beak"	
—parrot	Manganese deficiency	
—pliable	Rickets	Leg weakness also present
—undershot	Not indicative of disease	Normal feature following de-beaking
The Breast		
—blistered (cystic)	Breast blisters	Common, sometimes infected.
	Infectious synovitis	Affects broilers aged 4 to 8 weeks.
—crooked	Crooked breasts	Common. Genetic x nutrition x management defect
—emaciated	Cull	Evidence of chronic, non-specific illness
Comb		
—absent	Normal, following dubbing	
—bright red	Hyperaemia	Normal sign of good health
—congestion	Heart failure	
—cyanosis	Blackhead, Fowl cholera (acute) septicaemias generally	
	erysipelas, pullet disease	
	roundheart disease	
—gangrenous	Frostbite; ergot poisoning	In turkeys the snood is gangrenous
	local injury	in erysipelas
—pale	Anaemia	Often secondary. Also following internal haemorrhage
	Cull	
—scabby	Local injuries	
	Fowl pox	Mouth lesions usually present
—scurfy	Favus (whitecomb)	Parts of face also affected
—yellow	Jaundice; erythromyelosis	Isolated cases, usually in pullets
The Crop		
—impacted	Impaction of the crop,	Contents doughy and firm
—pendulous (turkey)	pendulous crop	Contents voluminous
—sour	Sour crop	Contents fluid and very acid
Ears		
	Mite infested	Head shaking occurs
	Middle ear abscess	Wry-neck
Eyes		
—discharging	Coryza; fowl pox	Lachrimation is otherwise uncommon in poultry
—purulent	Avitaminosis—A; Aspergillosis, etc.	
eyelids		
inflamed	Fowl pox; ammonia blindness	
scabby	blepharitis; deficiency of B vitamins	
cloudy	Local irritation	Keratitis
sticky	Vitamin A deficiency	Also occurs when no deficiency of Vitamin A present
swollen	Ammonia blindness	Old litter in use
cornea		
dry	Xerophthalmia	Avitaminosis-A
ulcerated	Kerato-conjunctivitis	Ammonia blindness associated with use of old litter
iris		
depigmented	Non-specific	Normal in young chickens
pupil		
dilated	Nicotine sulphate poisoning	Rare
distorted (Cat's eyes)	Genetic defect	Harmless
pinpoint	Fowl paralysis; irido-cyclitis	Blindness present

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
Eyes (contd.)		
lens		
opaque	Blindness (cataract)	Cause unknown; I.R.R. brooding sometimes related, also encephalomyelitis virus
Face		
—scurfy	Favus	Comb also involved
—swollen	Cellulitis	Local infection
(oedematous)	Coryza	Haemophilus infection of eyes and nose present
The Feathers		
—broken off	Genetic defect	Local injury
	Depluming mite infestation	Skin reddened
—curled	Frizzle feather	Genetic-nutritional defect
—dark specks amongst the vent and abdominal feathers	Northern fowl mite infestations	Readily confused with red mites
—depigmented (white)	Lysine deficiency	Seen in young turkey poults only
—loose	Botulism	Also non-specific feature
—matted with blood	Haemangiomata	Careful differentiation necessary.
	local injuries	
—matted with pale mucus	"Colds"	Neck and shoulders only
	Ammonia blindness	
—matted with white urates at the vent	Nephritis	"Pasted-up" vents common
	Salmonella infections	
—pecked	Feather pecking (vice)	Boredom an important feature
—ruffled	Coccidiosis, Spirochaetosis	
—vent feathers with white clusters of eggs	Lice infestations	Adult lice often visible
The Feet		
—"clutching hand"	Fowl paralysis	Of infra-red ray or genetic origins
—crooked toes	Crooked toes (chicks)	Pantothenic acid and biotin chiefly.
—dermatitis (poults)	B Vitamin deficiencies	Common. Local infection.
—swollen feet	Bumblefoot, Pasteurella abscesses	Uncommon. Urates present in joints
	Gout	
	Local injury	Management faults
—toes "balled up"	Non-specific	A vitamin B_2 deficiency symptom in young chicks
—toes curled under the feet	Curly toe disease	
—toes sloughed away	Local injury; Creosote poisoning	
	Biotin deficiency	Very rare
Head		
—anaemia (pale)	Caecal coccidiosis	A feature of many culls.
	Rupture of liver or aorta	Indicative of secondary anaemia, or internal haemorrhage
	Tuberculosis	
—congestion	Heart failure	Common non-specific feature
discoloured—bluey-green	Local injuries (bruising)	Associated with presence of subcutaneous haemorrhages
—purplish (cyanosis)	Blackhead, erysipelas, roundheart disease	
—shaking	Secondary to coryza or otitis	Normal feature in many fowls
sagging	Blindness	
swollen	Cellulitis; F. Plague	May follow careless implantation of sex hormone tablets
	Haemophilus influenza infection	
—tremors	Epidemic tremors	
—twitching or nodding	Newcastle disease	Sometimes sole symptom in turkeys
Infra-Orbital Sinuses		
—swollen	Coryza	Eyes discharging
	P.P.L.O. infections	
	Inf. sinusitis (turkeys)	Very common
	Avitaminosis—A; One-eyed roup	Eyes and throat also involved
The Legs		
—deformed hocks	Perosis, dislocation	Slipped tendon usually present
—discoloured hocks	Local injuries, ruptured tendons	
—swollen hocks	Hock disease (turkeys), rickets, arthritis (staphylococcal, atypical pullorum disease, chronic erysipelas)	Genetic x nutritional causes
	infectious synovitis	Other joints also swollen
—paralysed legs	Fowl paralysis	One or both legs affected
	Fractures	Only one leg usually affected
—gross thickening of the shanks	Osteopetrosis	Lameness often present
—raised scales (shanks)	Scaly leg disease	Becoming rare
—weakness	Rickets	

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
The Mouth		
—congested	"Blacktongue"	Nicotinic acid deficiency, rare
—gasping	Aspergillosis infectious laryngo-tracheitis gapes: acute Fowl Pest	Air hunger symptoms also arise from other causes, *e.g.* heatstroke
—pustular and diphtheritic lesions	Fowl pox	Lesions adherent (diphtheritic)
	coryza (P.P.L.O.)	Lesions easily removed
	avitaminous—A	Pharynx and oesophagus involved
—salivation	Poisoning	Rare
—scabs (angle of mouth)	Deficiency of B vitamins; local injuries	
The Neck		
—paralysed	Botulism (ducks); thiamine deficiency	Rare
	Newcastle disease	
—thrust forward	Folic acid deficiency	Turkey poults (rare)
—tremors	Epidemic tremors	Chicks aged 1 to 4 weeks
—twisted	Fowl paralysis; Newcastle disease; genetic defect (poults)	Wry-neck also occurs from other causes
Nose		
—discharging mucus	Coryza (colds), bronchitis, etc.	*H. gallinarum* or P.P.L.O. infections
—nares impacted with dried mucus	Roup	
The Nervous System		
—bicycling movements	Nutritional encephalomalacia	Also other causes
—coma	Non-specific	A sign of impending death
—convulsions	Nutritional encephalomalacia	Also pyridoxine deficiency.
	poisoning (netrophenide)	
—dullness	Non-specific	Sleepiness often precedes death.
		Extreme dullness "motionless" seen in nitrofurazone poisoning
—inco-ordination	Fowl paralysis; Newcastle Disease epidemic tremors, duck virus hepatitis, "poisoning", etc.	
—rigor	Fever; chilling; psittacosis	
—tremors	Epidemic tremors	Affecting chicks up to 5 weeks of age
—walking backwards	Nutritional encephalomalacia. Newcastle disease	
The Respiratory System		
—air hunger (gasping)	Aspergillosis	
	gapes; fowl paralysis	
	Infectious laryngo-tracheitis	
—"cheeping"	Salmonellosis, B.W.D.	A sign of discomfort or pain
—coughing	Infectious bronchitis; infectious laryngo-tracheitis; Newcastle disease	Blood coughed up with I.L.T.
—laboured breathing	Non-specific	Often related to hydropericardium, dropsy, etc.
—mouth breathing	Roup	"Pip"
—panting	Fever	Normal in hot weather
—rattling (throat)	Infectious bronchitis, Newcastle disease	Nervous symptoms often present Chicks frequently affected
—sneezing	Virus infections, P.P.L.O.	Adults frequently affected
—squawking	Foreign body in the larynx or trachea	
The Skin		
—bleeding	Haemangiomata (blood cysts)	Also local injuries
—blotchy (patchy)	Erysipelas (turkeys)	Snood gangrenous
—congested	Pullet disease, nephritis, egg peritonitis	Heart failure with venous stagnation present
—dehydration		Non-specific feature seen in hexamitiasis, pullet disease, etc. Also water starvation.
—discoloured	Bruising, local injury, haemorrhagic disease, vitamin K deficiency	Subcutaneous haemorrhages present
—eczematous	"Burns"	Rare in turkeys
—exfoliating	Dermatitis	Chemical irritation chief cause, also smothering
		Rare diathesis
—oedema	Salt poisoning	
	vitamin E deficiency	
	toxic fat disease	A new disease in Britain
—ulceration	Tumours (lymphomata)	
—yellow	Spirochaetosis; Erythromyelosis	Uncommon

STATE OF ORGAN	ASSOCIATED DISEASE OR CONDITION	COMMENTS
The Tail	F. paralysis or Genetic	Rare
Tongue		
—curled	Genetic defect	Common in turkey poults fed on dry mashes
—dehydrated	Secondary to roup	"Pip"
—discoloured	Black tongue	Nicotinic acid deficiency
The Voice	See text	
Wattles		
—absent	Normal following "cropping" operation	
—swollen	Secondary to coryza	Solid yellow pus present
—wrinkled and thickened	Fowl cholera (chronic)	Local Pasteurella infection Oedematous in F. Plague
The Wings		
—drooping	Intestinal coccidiosis blackhead, B.W.D. Normal in hot weather.	A debility feature seen also in other diseases.
—flapping	Fowl cholera (acute) roundheart disease	Often an impending sign of death
—paralysed	Fowl paralysis Local injury; Fractures and dislocations	One or both wings affected.

Coma is a terminal feature of many cases of poisoning, as well as being present when the diet is deficient in magnesium.

Inco-ordination (ataxia) is non-specific, although one type in chicks has been described which is of genetic origin. Ataxia with tetanic spasm (opisthotonic) has been recorded in association with magnesium deficiencies in chicks. Muscular weakness resulting in inco-ordination is commonplace, because sick poultry do not eat. This may be because they are physically unable to reach the feed troughs or because of bullying.

Paddling of the feet or bicycling movements and ataxia generally may be noted in crazy chick disease and also in the terminal stages of toxic fat disease.

A flaccid paralysis of the neck is seen in Botulism, some cases of fowl paralysis, and supposedly also in tapeworm infestations. Cervical paralysis in poults may be of Folic Acid deficiency origin.

The rigidity of the carcase once death has supervened may be characteristic, as for example in the incidence of opisthotonus seen in some cases of turkey "X" disease; a feature which may sometimes also be seen immediately prior to death.

Rigor is a feature of chilling and it may also be seen in particular diseases, *e.g.* psittacosis.

Stumbling and falling over are often observed in epidemic tremors and crazy chick disease; also in cases of duck virus hepatitis. "Stargazing" is seen in vitamin B deficiency, due to paralysis of the anterior neck muscles.

Torticollis (wry-neck) occurs in cases of fowl paralysis when the associated nerves are infiltrated, abscesses of the middle ear, etc. In the turkey spurious torticollis is linked with an arthritic condition of the vertebrae, being of genetic origin and first noticed when the poult is 2 to 3 weeks old.

Trembling is an obvious feature in epidemic tremors, but loss of muscular tonus is also seen in various toxic states. Incidentally, a rapid vibration of the head and neck in poults may also be of genetic origin, being related to a single sex-linked recessive gene. Twitchings may indicate Newcastle disease in chickens, in turkeys a nodding of the head is of equal significance.

Vertigo and convulsions are seen in chicks when there is a deficiency of magnesium in the diet, in nutritional encephalomalacia, epidemic tremors, some cases of Newcastle disease and poisoning by nitrofurazone. Conversely extreme dullness is seen in cases of nitrophenide poisoning.

Walking backwards may be a feature of Newcastle disease, but it is seen also in crazy chick disease. Avian encephalomyelitis of equine virus origin is stated to cause staggering.

References

BIESTER & SCHWARTE (1959). "Diseases of Poultry." Iowa State University Press, U.S.A.

BLOUNT, W. P. (1949). "Diseases of Poultry." Baillière, Tindall & Cox, London.

———. (1955). *Vet. Rec.* **67.** 1,087.

CHU, H. P. (1960). "Laboratory Handbook on Diagnosis of Poultry Diseases." FAO Monograph No. 2.

EWING, R. (1951). "Poultry Nutrition." South Pasadena, California, U.S.A.

GORDON, R. F. (1956). *Vet. Rec.* **68.** 547.

———. (1961). Report on Avian Respiratory Disease in Gt. Britain. Office Int. des. Epizooties. No. 597.

GRAU, C. R. (1945). *Proc. Soc. Exp. Biol.* **59.** 177.

HEYWANG, B. W. (1940). *Poult. Sci.* **19.** 201.

MEDWAY & KARE (1959). *Ibid.* **38.** 636.

MERCK, SHARPE & DOHME INTERNATIONAL (1958). "Poultry Service Manual." U.S.A. Poultry Diseases under Modern Management. *P. World*, 1961. Dorset House, London, S.E.1.

PRIER, J. E. (1953). "Turkey Diseases." Interstate Printers & Publishers, Danville, Illinois, U.S.A.

SCHMITTLE, *et al.* (1958). *J.A.V.M.A.* **132.** 216.

SHANNON, J. L. (1947). Personal communication.

STURKIE, PAUL D. (1954). "Avian Physiology." Comstock Publishing Associates, Ithaca, New York.

WARREN, D. C. (1937). *Kansas St. Ag. Exp. Bull.* No. 44.

WHEELER, *et al.* (1957). *Poult. Sci.* **36.** 1,167.

2.—The Control and Prevention of Poultry Disease

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SUMMARY.—The control and prevention of poultry disease is discussed under 5 main headings: General management, including reference to non-specific chick mortality, egg-borne infections, parasitic, respiratory and nutritional conditions; genetics and disease; control of disease by legislation; use of vaccines; and use of drugs and antibiotics. In the last section an attempt is made to present in tabular form the information available concerning proprietary preparations in common use to-day for the prophylaxis and therapy of poultry diseases.

THE diagnosis of disease among poultry should not present any difficulty to the qualified veterinarian once he has become familiar with the husbandry and management of domestic poultry under modern conditions. The ability to distinguish the significant from a mass of irrelevant detail, highly developed powers of observation, together with an almost intuitive assessment of the evidence available, are the stocks in trade of the successful diagnostician in whatever field he is working. But the control and treatment of poultry disease calls for a different approach, and the average veterinarian may find it difficult at first to adjust his thinking to the changed circumstances of his patients. No longer is he concerned with administering treatment to an individual and in assessing its effect often over a period of days. The fate of the individual fowl, turkey or duck is of little importance, and the success of treatment and advice is measured by a decrease in mortality and severity of flock symptoms, and by improvement in food consumption or egg production. Only rarely is a veterinarian required to estimate the dose of an antibiotic or drug for an individual bird; he must, however, be familiar with the relative merits of the preparations available for the treatment of disease in poultry, and know at what levels and concentrations they should be incorporated in the food or water of a flock requiring treatment. Furthermore, in few other fields does management and environment of the flock exert so fundamental an effect on the course of disease, and the veterinarian without experience of poultry problems may well waste his client's money and do nothing to enhance his own reputation among the poultry community unless he appreciates this essential point.

The purpose of this paper is to discuss control of poultry diseases in general terms rather than to attempt to provide a practitioner's guide to treatment of disease. Where applicable specific measures will be mentioned, but to be successful in controlling disease among the poultry flocks of his clients, the veterinarian must be prepared to tackle the problems he meets on the broadest possible basis.

A. General Management and Disease

1. *Non-specific Chick Mortality*

The toll of mortality due to mismanagement or adverse environmental conditions is probably greatest among young birds during the first vital four weeks of life. Not only is more than 60 per cent. of the annual mortality during this period attributable to factors other than specific disease, but checks and setbacks to normal progress as a result of adverse environmental conditions undoubtedly affect the subsequent development and profitability of the birds whether they are destined finally for table purposes, egg production or potential breeding stock.

The veterinarian investigating baby chick mortality has something in common with a detective on a crime inquiry. Both investigators are faced with a number of facts, many of them irrelevant and often misleading. The veterinarian will exclude the possible presence of infectious diseases as automatically as the detective looks for finger prints, but at the same time he must be careful not to overlook other more common causes of chick mortality.

He should remember that the rearability of baby chicks may be affected by the conditions existing during incubation and hatching, by the manner in which they are handled at the hatchery prior to despatch and by the delays and accidents to which they may be subjected during transit to the rearing farm. A check or setback during the first day or two after hatching may result in losses in transit, and in failure of many individuals to settle down and start feeding normally.

Uneven temperatures, draughts and overcrowding under the brooders will increase the slight mortality which is often inevitable during the first day or two of rearing. The term "chilling," so freely and loosely used in laboratory reports, is misleading and is best avoided because baby chicks are comparatively resistant to quite low temperatures, but on the other hand they cannot stand overheating, particularly when associated with overcrowding.

Failure to start feeding normally, the condition known as "starve outs" among turkey rearers, characterised by the completely empty gizzard and intestines of poults dying when about 2 to 3 days old, may be the sequel to a set-back due to one of the factors mentioned, but a similar picture sometimes results from an inadequacy of trough space or to feed and water containers not being readily accessible to the baby chicks.

2. *Transmission of Egg-borne Infection*

A high standard of management and hygiene is essential both on supply farms and at hatcheries in order to prevent the introduction and spread of infec-

tious diseases, some of which may result from shell contamination during or soon after laying and subsequent penetration into the yolk through the shell pores. The day-to-day routine in a modern hatchery presents countless opportunities for the spread and dissemination of bacterial, virus and fungal diseases and unless the rules laid down for the prevention and control of disease in hatcheries are rigidly adhered to serious outbreaks are liable to occur.

Naturally clean hatching eggs from the supply farms should be the aim of all hatchery managers and an efficient method of dry-cleaning dirty shells should be available at the hatchery. Bi-weekly introduction of clean nest-box litter and more frequent collection of eggs may prove cheaper than the purchase of expensive egg-washing machines or construction of fumigation chambers. Routine fumigation at the hatchery of all eggs prior to setting in the incubators and regular cleaning and fumigation of the machines after each hatch is taken off are routine essentials. Regular use of bactericidal aerosols are popular in some hatcheries as an adjunct to the manual cleaning.

3. *Parasitic Disease*

Outbreaks of coccidiosis, blackhead and certain worm infestations can be prevented by relatively simple measures, adapting knowledge of the life history of the parasites to management on the farm. The life cycle of coccidia is interrupted by the moving of fold units every other day; blackhead in turkeys can be checked by avoiding all contact between young turkeys and adult fowls and by rearing in wire floored brooders and verandas; gape-worm outbreaks (syngamiasis) are almost unheard of among chicks reared away from a broody hen and without contact with earthworms.

Ground used for rearing should be rested after the young stock have been moved into their permanent laying or breeding quarters, and adult birds should *never* be allowed on it from one season to the next. In this way the level of parasitic infection on the ground is kept to a minimum, but at the same time leaves sufficient present on the average farm to stimulate immunity in susceptible birds without causing disease.

Under broiler conditions control of coccidiosis would be impossible without resort to the use of coccidiostatic drugs, but nevertheless the presence of a drug in a broiler feed does not always prevent a build-up of infection with consequent outbreaks of coccidiosis, unless specific measures are taken to avoid it. In between one crop and another the litter should be heaped and allowed to heat. Litter in both broiler houses and in deep litter laying houses must be dry and friable to permit maximum fermentation with resulting destruction of coccidial oocysts and worm eggs. Efficient litter control is impossible in houses which are not properly insulated and ventilated.

4. *Respiratory Disease*

All respiratory diseases are infectious to some extent but even the mildest outbreak of colds may become a serious debilitating chronic condition in a flock kept in a badly ventilated or overcrowded house. Furthermore the rapidity of spread and mortality from the more serious diseases is greatly enhanced under such conditions. Recurrent outbreaks of coli septicaemia and chronic respiratory disease in one crop after another are not uncommon in some broiler houses and resort to drugs and antibiotics prove useless until predisposing causes such as faulty ventilation, lack of insulation or possible overcrowding have been attended to.

Even with the more natural rearing of young stock in night arks and fold units, inadequate ventilation may be a factor in checking growth and encouraging spread of respiratory disease. Common faults are the keeping of too many birds in slatted floor houses, thus cutting down intake of air from the floor, possibly already reduced by long grass growing up round the sides of the unit. Under free range conditions, overnight overcrowding is not uncommon due to birds exercising a preference for those houses situated nearest to the entrance of the field by which the food cart appears.

5. *Layout and Siting of Buildings in Relation to Disease Control*

It has long been realised that it is bad husbandry to allow different age groups of poultry to mix freely with one another. But the risk of spread of disease by airborne infection from one flock to another and from farm to farm has only been appreciated in recent years. The danger is greatest with the respiratory virus diseases, a fact which is believed to have been of considerable significance during recent fowl pest epidemics, particularly in the densely poultry populated parts of Lancashire and in parts of East Anglia and Sussex where broiler production is concentrated on a large scale, resulting in a much greater volume of potentially infected dust and debris becoming airborne from infected premises.

The risk of disease spreading in this way could sometimes be lessened by more careful planning and siting of houses in relation to the prevailing winds. The filtering or disinfection of air as it leaves an intensive poultry unit presents many practical difficulties but it is not too much to hope that collaboration between engineers and chemists may eventually produce an answer to this problem in the future.

The importance of placing brooder houses as far away as possible from adult fowls in order to reduce the degree of exposure experienced by baby chicks to the agents causing leucosis and fowl paralysis is now generally realised. The incidence of these diseases developing subsequently among growers and adult fowls has proved on many occasions to be much reduced, if the importance of rearing the baby chicks in isolation and as far away as possible from adult fowls has been appreciated and acted upon.

6. *Nutrition and Disease*

This specialised aspect of management in relation to prevention of disease was covered adequately by Gibson (1960) in his paper to the Glasgow Congress. Circumstantial evidence accumulates every day

emphasising the complicated aetiology of certain common nutritional diseases. On many occasions it is impossible for the diagnostician to explain the occurrence of symptoms and lesions of deficiency disease in baby chicks and turkey poults receiving what on investigation proves to be an adequate diet; or when disease occurs among a comparatively small number of individual birds with the majority of a batch, amounting to several thousands, rearing and growing satisfactorily. Nutrition of the breeding flock and the genetical make up of individual birds, variations in vitamin and mineral requirements of individuals and in their ability to absorb and utilise them would all seem to play a part in some outbreaks.

Stock owners are frequently over-quick to blame the proprietary feed manufacturers, when in fact the failure of their flocks or herds to rear, grow or produce satisfactorily is in reality often due to their own indifferent management or to the presence of infectious disease. Most veterinarians have been tackled at one time or another by food firm representatives as a result of a client's determination to make the most of some chance observation concerning the nature or suitability of a particular foodstuff. The position of the veterinary civil servant is even more difficult when his remarks are liable to be quoted back, as "the Ministry says," and the repercussions following a loosely expressed opinion are sometimes far-reaching. Nevertheless, the first responsibility of a veterinarian is to the poultry owner, and if his assessment of a particular problem leads him to suspect that the food could be a factor in causing disease, it is his duty to warn the owner accordingly. On poultry farms to-day, the number of individual birds liable to be rendered uneconomic, should the food be unsuitable or contain some toxic substance, is so great that a delay of a few days is often of importance, and time does not a permit a full investigation before a change of food is recommended. On many occasions the improvement in condition of a broiler flock, which has not been making satisfactory progress, or of a laying flock with poor production, following a change of food, is as helpful in establishing a diagnosis, as is the response of the individual cat, dog or cow to the empirical injection of a drug or antibiotic. Only after such a demonstration should there be any talk of collecting food samples for analysis and the lodging of complaints to the local food merchant or representative.

B. Selective Breeding and Control of Disease

The specialist geneticist is now a member of the team on many large poultry breeding establishments. Having proved the value of his contribution to the industry in the developing hybrid strains of poultry which are proving economically more profitable under modern conditions than those based mainly on selection of breed characteristics and conformation, the geneticist may soon be called upon to apply his experience in the field of disease.

It seems possible that in concentrating on selection for maximum egg production, rapid growth rate, high fertility, and hatchability, certain undesirable traits have "nicked" in the strains under test, which might explain the unexpected non-specific mortalities and disappointing rearing results experienced from time to time among baby chicks and poults of a particular strain or cross.

Selection for resistance to disease is inherent in all progeny testing, although the results are based on overall mortality in the group rather than to the demonstration of resistance to a specific condition. The achievements of the Cornell and East Lansing workers in America in relation to fowl paralysis and leucosis provide an example of the progress possible when selective methods are applied to the control of a specific group of diseases.

C. Control of Poultry Disease by the State

1. Under the *Diseases of Animals Act* (1950) the Minister is empowered to make Orders for the control of certain named poultry diseases. These powers have been used so far in respect of:

(1) *Fowl Pest* (including Newcastle disease and fowl plague).

(2) *Virus Hepatitis* of ducklings and

(3) *Psittacosis or Ornithosis*

These diseases are not notifiable, but powers to slaughter, detain, isolate, and to require cleansing and disinfection exist under the Act and the relative Orders.

For virus hepatitis a live virus vaccine, developed at Weybridge (Asplin, 1958), is in use on an experimental basis in parts of Norfolk and Suffolk, but only in certain circumstances; it is not available in other parts of the country.

The other diseases which are named in the Act are—

Pneumo-encephalitis (identified as Newcastle disease after passing of the Act).
Bacillary white diarrhoea
Fowl typhoid
Fowl pox
Fowl paralysis
Fowl cholera
Contagious bronchitis
Infectious laryngotracheitis

The Minister may also extend the list or exclude any of the diseases mentioned.

2. Newcastle disease and fowl plague are the only diseases of poultry notifiable under the *Diseases of Animals Act* (1950). Fowl plague has not been diagnosed in this country since the early 1920's but Newcastle disease was reintroduced in 1947 and has probably been present ever since. The policy of the Minister of Agriculture and Fisheries under the Fowl Pest Order 1936, is the eradication of Newcastle disease by compulsory slaughter of stock in confirmed outbreaks with payment of compensation to the owners. This policy resulted in the prompt eradication of the acute "Asiatic" form of the disease and has been equally successful in controlling the less acute "American" type in most parts of the country. At different times and in fairly well defined areas, for example Lancashire in 1955-8 and the Eastern Counties 1958-61, and in spite of the measures taken, large

numbers of outbreaks have occurred. The relative freedom from infection in Lancashire since 1958 may, however, be taken as a measure of the success that can attend the policy, even in an area where infection was previously very heavy. These periodic epidemics can frequently be linked with the activities of dealers, slaughtermen and the like, and more recently with intensive methods of husbandry involving forced draught ventilation which increases the chances both of introducing airborne infections and of disseminating them more widely. The cost of slaughter with compensation has risen with the development of "intensivism" and the great increase in the size of poultry flocks.

In 1960, the Minister appointed a Committee of Enquiry under the Chairmanship of Sir Arnold Plant to "review the policy and arrangements for dealing with fowl pest in Great Britain and to advise whether there should be any changes made in the light of the growth of the industry, present scientific knowledge and technical and administrative experience gained in recent years in this and other countries."

3. *The Agriculture Act of* 1937 empowered the Minister to introduce a Poultry Stock Improvement Plan, a measure which included proposals for disease control as well as stock improvement. Membership of this plan has been entirely voluntary and unlike most other programmes for the control or eradication of an animal disease, it carries no direct financial inducement other than free veterinary services. From the first introduction of this plan, incorporating the Accredited Poultry Breeding Stations and Accredited Hatcheries Schemes, the poultry industry has co-operated most loyally and resolutely with the Ministry in making it a success, and as a result the crippling lossess from *Salmonella* egg-borne disease and hatchery-transmitted infections which might otherwise have checked post-war expansion of the industry have largely been avoided. Furthermore, it can be claimed that the Schemes have also played a significant part in safeguarding public health through checking spread and reducing the amount of *Salmonella* infection in poultry.

The measures at present embodied in the disease control provisions of the P.S.I.P. have led to a considerable reduction in the incidence of pullorum disease; twenty years ago (1941), 23 per cent. of the chick batches sent to the Central Laboratory, Weybridge, for diagnosis, proved to be infected with pullorum disease, while 41 per cent. of the breeding flocks blood tested that year contained reactors to the agglutination test; ten years ago, these figures were 9.8 per cent. and 15 per cent. respectively; in 1960, they were 3.4 per cent. and 7 per cent. For the past few years, however, little progress has been made and the 1960 figures showed only slight improvement over those of the previous 4 to 5 years. It must, however, be emphasised that the progress made so far has been achieved within the confines of a voluntary scheme without any direct financial reward. It may be that a more widely based Poultry Disease Control Scheme available to all owners of poultry breeding stock will be necessary before final eradication of this disease can be contemplated.

D. The Use of Vaccines in the Control of Poultry Disease

Only 3 vaccines are available commercially in this country for the control of diseases of poultry.

1. *Fowl Pox*—A live pigeon pox virus scab vaccine is supplied from the Central Veterinary Laboratory, Ministry of Agriculture, Fisheries and Food, Weybridge, at 1s. 9d. for 30 doses. This vaccine, applied by scarification of the skin on the upper part of the leg, conveys a satisfactory immunity against fowl-pox infection for up to 6 months.

2. *Fowl Typhoid*—A vaccine, consisting of a live rough strain of *Salmonella gallinarum* (known as 9R), is supplied by the Animal Health Trust, Biologicals Division, Kennett, Newmarket, at 6d. per dose. The immunity conferred by this vaccine lasts from 4 to 6 months.

With the exception of fowl pox vaccine, the use of live vaccines, on flocks accredited under the P.S.I.P. is not allowed without prior authority of the Ministry.

3. *Swine Erysipelas — Aluminium hydroxide* absorbed vaccines prepared for the prevention of erysipelas in pigs are also suitable for the vaccination of turkeys. These vaccines* are not normally used unless the disease has been diagnosed in a flock and is causing daily loss among adult birds.

The wider use of vaccines in this country is a matter of some controversy at the present time. The manufacture for sale or the importation for sale of sera, vaccines and certain other biological therapeutic substances intended for use in animals, including poultry, is controlled under the Diseases of Animals Act through the Therapeutic Substances Order (1952) as amended. A licence cannot be granted in respect of any therapeutic substance the use of which, in the opinion of the Minister, may spread any disease which is a disease for all or any of the purposes of the Act. Thus the manufacture for sale of live vaccines for the control of *e.g.* fowl pest, infectious bronchitis, infectious laryngotracheitis and encephalomyelitis (epidemic tremor) cannot take place as the law now stands.

Live vaccines, if egg adapted, may possibly transmit other infections; also their use for various respiratory diseases may act as a stress factor precipitating clinical disease from other existing latent infections. Dead vaccines are unlikely to be responsible for the transmission of other infections or to aggravate other diseases if present in a vaccinated flock, but they all share the disadvantage of having to be administered by individual injection. The Committee, to which reference has already been made, will no doubt be discussing the application of these broad considerations as they apply to fowl pest.

E. Drugs and Antibiotics in the Control of Disease

Outbreaks of disease in poultry are influenced by so many different factors that categorical recommendations concerning the efficacy of the many proprietary preparations advertised and sold for the treat-

* Erysilin, Glaxo Ltd. Duovax, Cynamid Ltd.

ment and prevention of poultry diseases, might be misleading. The maximum value from drugs and antibiotics can only be obtained if they are used sparingly and intelligently.

The veterinarian should not prescribe medication of a flock until he has carefully assessed all the circumstances which might have contributed towards an outbreak of disease, together with the economic

TABLE I

DRUGS AND ANTIBIOTICS IN COMMON USE IN POULTRY PRACTICE

Disease	Trade name	Manufacturer	Active principle	Dose and method of use	Remarks
Blackhead	Entramin "A" (22½% premix)	May & Baker	Acetamidonitro-thiazole	**Proph.** 2 oz. premix per cwt. food (0·025%) fed continuously. **Therap.** Double strength for 7–14 days followed by 2 oz. per cwt. continuously. **Proph.** ½ fl.oz. per 2 gallons drinking water.	100 gm. capsules available for individual
	Entramin (16% solution	,,	2 amino-5-dinitro-thiazole	**Therap.** ½ fl. oz. per gallon water	May affect fertility in older birds if fed continuously.
	Hepzide	Merck	Nithiazide	**Proph.** 0·025% fed continuously soluble form in drinking water 1 oz. per gallon	Give at preventive level after treatment
	Turk-E-San	Boots	Sodium acetarsol	One tablet to 2½ gallons water, given continuously	Discontinue 7 days before killing.
	Neftin	Smith, Kline & French	Furazolidone (4·4% premix)	0·01%–0·02% fed continuously 0·04% for established infections for 10–14 days	
Blepharitis in turkeys	Hydromycin ear-eye ointment 1·5%	Boots	Hydrocortisone acetate and neomycin sulphate	**Therap.** Apply daily into the conjunctival sac	
	Chloromycetin-ophthalmic ointment 1%	Parke-Davis	Chloramphenicol 1%	,,	
Coccidiosis	Sulphamezathine 16% soln.	I.C.I.	Sulphadimidine	**Therap.** 0·02% in the drinking water (2 fl. oz. of 16% soln. per gallon) 3 days on—2 off—2 on. Intestinal cocci—use ½ strength but for an additional period of three days *i.e.* 3 on—2 off—3 on—2 off—3 on.	Use earthenware or new galvanised vessels.
	Embazin 10·32% soln. 22·5% premix	May & Baker	Sulphaquinoxaline	**Proph.** 0·0125% in the food. 1 oz. premix to 1 cwt. food. (0–8 weeks) **Therap.** 4 oz. premix to 1 cwt. food (for 7 days). 0·04% in the water (2 fl. oz. of 10·32% soln. to 3 gallons drinking water) 3—2—3.	Sulphaquinoxaline more suitable for ducks, turkeys and pheasants as less unpalatable.
	Nefco	Smith, Kline & French	Nitrofurazone	**Proph.** 0·01% fed continuously	Continuous feeding for more than 10 weeks not recommended. Pheasant chicks sometimes show marked susceptibility to toxic effects.
	Chixin	I.C.I.	,,	**Therap.** 0·02% fed for 7 days	
	N.F.Z.	Coopers	,,	Water soluble form available.	
	Nicoxin	M. & B.	Nicarbazin	**Proph.** only.	Not recommended for turkeys or adult fowls. Causes depigmentation of coloured eggs.
	Nicrazin	Merck	,,	Premix (0·01–0·015%) fed up to 16 weeks	
	Bifuran	Smith, Kline & French	(0·0055% Nitrofurazone & (0·0008%) furazolidone mixed	**Proph.** Feed continuously ½ lb. per ton food. **Therap.** 2 lb. per ton for 7 days.	Reputed to improve food conversion. Soluble tablets available for addition to water as a treatment.
	Zoamix (25% premix)	Dow	Zoalene	**Proph.** feed continuously 0·0125% (18 oz. premix per ton)	
	Trithiadol	Bayer	Bithional & Methiotriazemine	**Proph.** Feed continuously 0·0125% (2 lb. per ton)	
	Amprolmix (22·5% premix)	Merck	Amprolium	**Proph.** Feed continuously at 0·0125% (1¼ lb. per ton)	

TABLE—*continued*

Disease	Trade name	Manufacturer	Active Principle	Dose and method of use	Remarks
	Whitsyn S	Whitmoyer & Reed Ltd.	Sulphaquinoxaline and pyrimethamine	**Therap.** Soluble form for administration in drinking water.	Synergistic effect of pyrimethamine allows use of sulphonamide at lower concentration than that normally needed to control disease ; no risk of toxicity.
	Whitsyn 10	,,	,, ,,	**Proph.** Premix for continuous feeding .	
Colisepticaemia	Neftin	Smith, Kline & French	Furazolidone	0·01% in the food for 10 days.	Use according to results of sensitivity tests on cultures isolated from outbreak.
	Terramycin	Pfizer	Oxytetracycline	100–200 gm. per ton in the food	
	Aureomycin	Cyanamid	Chlortetracycline		
Coryza & CRD (and secondary respiratory infections)	Avisol	M. & B.	Sulphathiazole (40% soln.)	**Therap.** 2 fl. oz. to each gallon drinking water. Individual birds —$\frac{1}{4}$–$\frac{1}{2}$ teaspoonful twice daily for 2–3 days.	
	Terramycin A.F. Sol. powder	as above	Oxytetracycline	4–5 oz. per 10 gallons water or 100 lb. food for 5–7 days.	Continue for further 7 days at half dosage.
	Terramycin in oil	Pfizer	Oxytetracyline	$\frac{1}{2}$–2 c.c. inj. s/c	
	Streptomycin	Glaxo	Streptomycin sulphate	25 mg. per kg. intramuscularly.	
	Aureomycin	Cyanamid	Chlortetracycline	100–200 gm. level in food or equivalent in water	
	Vet-Sana	E. R. Squibb & Sons Ltd.	Procaine Penicillin G + Dihydrostreptomycin sulphate	0·21 c.c. per lb. bodyweight	0·21 c.c. = 1 dose with Vet-Jecta gun.
	N–S dust	Whitmoyer & Reed Ltd.	Dihydrostreptomycin sulphate and neomycin sulphate.	**Therap.** Micronised dust applied over birds with a gun.	Reduced mortality and increased weight gains claimed. Estimated cost $\frac{3}{4}$d. per bird.
Duck Septicaemia	Sulphamezathine	I.C.I.	Sulphadimidine	**Therap.** 0·1% soln. in drinking water (2—3—2)	Some danger of toxicity.
	Aureomycin	Cyanamid	Chlortetracycline	100 gm. per ton for 5 days or equivalent in water.	
Ectoparasites	Louse powder	Coopers	Gamma B.H.C.	Used as a dust	
	Gammexane	I.C.I.	(0·5% gamma benzene hexachloride)	,, ,, ,,	
	Poultry perch paint	Coopers	Gamma B.H.C.	Apply to perches and woodwork or legs of battery birds	Red mite and northern mite.
	Poultry Aerosol	Coopers	Pybuthrin	Used as aerosol	Red mite and northern mite. Can be used on turkey poults.
	Malathion	Cyanamid		Aerosol or spray	Red mite and northern mite.
	Sevin	Boots Ltd.	1-naphthyl-N-methyl carbamate	5% dust or 0·25% spray.	Effective against mites, lice and fleas.
Erysipelas (turkeys)	Crystapen	Glaxo	Crystalline Sodium penicillin g	**Therap.** 100,000 units i/m	
	Benapen	,,	Benethamine penicillin	1 injection of (160,000 units) should suffice	
Infectious Sinusitis	Streptomycin	Glaxo	Streptomycin sulphate	**Therap.** 150–250 mg. i/m or in dewlap 100–150 mg. dissolved in 1 or 2 c.c. water and injected into each sinus.	Some danger of toxicity if overdosed.
	Terramycin in oil	Pfizer	Oxytetracycline	2–5 c.c. s/c or $\frac{1}{4}$–$\frac{1}{2}$ c.c. in each sinus. Injected into sinuses after withdrawal of pus.	Drain sinuses if distended.
	Terramycin A.F.	,,	,,	2–3 oz. per 10 gallons water or 100 lb. food.	
	Crystamycin	Glaxo	Sod. penicillin + streptomycin	**Therap.** 100,000 units plus 0·15–0·25 gm. per bird	
	4% silver nitrate 15% mild silver proteinate	B.P.		Injected into sinuses after examination of pus	

TABLE—*continued*

Disease	Trade name	Manufacturer	Active principle	Dose and method of use	Remarks
Sinusitis (air sac type)	Crystapen and Streptomycin	Glaxo	Sod. penicillin g. Streptomycin sulphate	200 mg. streptomycin and 100,000 units penicillin and 1 c.c. dist. water i/m daily for 3 days.	
Infectious Synovitis	Aureomycin	Cyanamid	Chlortetracycline	**Therap.** 200 gm. per ton of food or equivalent in water. Lower dosage if combined with terephthalic acid or fed with reduced calcium content in the ration.	Must be given continuously until slaughter.
	Terramycin A.F.	Pfizer	Oxytetracycline		
	Neftin	Smith. Kline & French	Furazolidone	0·04% in food.	Less effective.
Moniliasis	Mycostatin	E. R. Squibb & Sons Ltd.	Nystatin	Mycostatin 20–feed supplement 2½–5 lb. per ton for 7–10 days.	Equivalent to 50–100 gm. mycostatin per ton.
Pasteurellosis (turkeys and fowls)	Embazin	M. & B.	Sulphaquinoxaline 10·32% soln.	**Therap.** 2 fl. oz. per 3 gallons water (0·04%) or 1 fl. oz. per 3 gallons for 5—7 days.	3—2—3 basis.
	Terramycin in oil	Pfizer	Oxytetracycline	**Poultry** 1–4 c.c. according to wt. subcutaneously. **Turkeys** 2–6 c.c. (large birds) or 0·25 c.c. intravenously	Change ground where possible after treatment particularly where turkeys are affected.
	Terramycin A.F. soluble	,,	,,	3–4 oz. per 10 gallon water or in food.	
	Streptomycin	Glaxo	Streptomycin sulphate	**Turkeys** 25 mg. per lb. i/m or s/c	In poults — MLD during 1st week is approx. 0·83 mg. per gm. Keep low dosage and make a few pilot injections.
	Crystamycin	Glaxo	Sodium penicillin & streptomycin.	50–100,000 units plus 0·15–0·25 gm. per bird	
	Crystapen	Glaxo	Crystalline sodium penicillin g.	25,000–100,000 I.U. in 2 c.c. water i/m	Must be repeated.
	Intramycetin	Parke Davis	Chloromycetin 15% aqueous soln.	0·5 c.c. i/m	
Pullet disease	Aureomycin	Cyanamid	Chlortetracycline	**Therap.** 100 gm. level in food or equivalent in drinking water	
	Terramycin A.F. sol.	Pfizer	Oxytetracycline	4 oz. per 10 gallons water or 100 lb. food for 5–7 days.	
	Epsom salts	B.P.	Mag. sulp.	1 lb. per 100 birds in mash.	
	Molasses			10% molasses in drinking water or 10–20% in wet mash.	
Pullorum disease Salmonellosis and fowl typhoid	Neftin	Smith. Kline & French	Furazolidone (4·4% premix)	**Proph.** 0·01–0·015% continuously **Therap.** 0·04% in food for 10 days. Individual birds 50 mg. tablet for 3 days followed by medicated mash.	In Fowl Typhoid—remove to fresh ground after treatment and repeat treatment. 0·011% delays sexual maturity in male birds therefore continuous feeding inadvisable for stock cockerels. Pheasants, turkeys, ducks } avoid any risk of over dosing
Ruptured aorta in turkeys	Serpasil	C.I.B.A.	Reserpine	**Proph.** 0·5 mg. per lb. food for 5 days followed by 0·1 mg. per lb. food for 2 weeks.	Reserpine is available on prescription in U.K.
Staphylococcal arthritis	Crystapen	Glaxo	Sod. penicillin g.	**Therap.** 25,000–100,000 I.U. in 2 c.c. i/m	Must be repeated.
	Terramycin	Pfizer	Oxytetracycline	1–4 c.c. subcutaneously.	
	Vet-Sana	E. R. Squibb & Sons Ltd.	Procaine penicillin g. and dihydrostreptomycin in oil	0·21 c.c. per 5 lb. bodyweight	Used with Vet-Jecta implanter 1 shot = 0·21 c.c.

TABLE—*continued*

Worm infestations	Phenos	Boots	Phenothiazine	**Therap.** 2/4 oz. per 100 birds in food for 1 day only: repeat after 6–8 weeks.	Controls caecal worms thus preventing blackhead. Do not give birds under 5 weeks or any with blackhead disease.
	Phenovis	I.C.I.		1 lb. sufficient for 900 fowls (450 turkeys) (adults)	
	Coopane	Coopers	Piperazine	Give as directed by manufacturers for 1 day.	Effective vermifuge against round-worms. Suitable for all poultry over 8 weeks.
	Pipricide	Burroughs, Wellcome			
	Safersan	Boots			
	Helmox	I.C.I.	Piperazine	Dissolve and scatter over food as directed	On trial for removal of gapeworms. Unsuitable for pheasants.
	Dicestal	M. & B.	Dichlorophen	One or two tablets by mouth, repeat in 28 days.	For removal of tapeworms.
Gizzard worms (geese)	Carbon tetrachloride	I.C.I./Boots	CCl_4	2 c.c. per kilo bodyweight. Given in capsule *per os*.	

factors operating on each occasion. Eight to ten thousand broiler chickens, for instance, consume a ton of food every day after about the 8th week and the cost of medication and its likely effect must be judged alongside the period of time left before the flock is to be killed.

The results following treatment of poultry flocks with drugs and antibiotics are bound to differ widely from one outbreak to another, but as this paper is written primarily for the guidance of the veterinary surgeon in practice, an attempt has been made to present, as a Table, details concerning most of the proprietary remedies available to-day for the treatment or prevention of disease. It is hoped that all preparations of proved value are included in the Table, but no claim is made for its completeness; it is probable that additional items could be added to it, if not to-day, certainly in a few months' time.

Acknowledgments.—The author is indebted to his colleague Mr. B. S. Hanson, M.R.C.V.S., for the preparation of the Table concerning drugs and antibiotics in common use in poultry practice.

References

ASPLIN, F. D. (1958). *Vet. Rec.* **70**. 1,226.
GIBSON, W. W. C. (1960). *Ibid.* **72**. 971.

THE SPEAKERS' INTRODUCTION

The Chairman, Mr. W. E. Beattie, said that he would start by introducing himself: to many people present, his was just one of the names in the Register. It had been there for 14 years. He had spent a little time in practice and further time with the Ministry of Agriculture. Then a certain poultry firm in the north of England had approached him saying that they required the services of a veterinary surgeon. That was nearly 5 years ago. Since then, he had specialised in poultry work, trying to get to know some of the poultry diseases and the industry.

As a guide to the direction of the industry to-day, his firm manufacture some 20,000,000 day-old chicks per year. A new laboratory was just being built at a cost of between £15,000 and £20,000, so, from the veterinary angle, it was thought that there was some future and some importance in disease work in poultry.

There was a statistical section, and the firm had an electronic computer. The head statistician was a Doctor of Philosophy and the head geneticist was a Doctor of Philosophy; so also was the nutritionist. It would, therefore, be seen that the poultry industry was moving forward.

Figures which had been published in January of this year might startle some of the audience. It was extremely difficult to get estimates of the agricultural industry in this country. Agricultural returns were not necessarily factual, for various reasons, but statistics had been compiled, and it might surprise the audience to know that in 1937-38 there was £300,000,000 worth of poultry and poultry produce in this country: in 1959-60 there was £1,493,000,000 worth. Therefore, even though the profession might not yet be taking what could be termed a really serious interest, agriculture itself was, and the Government was.

Turning to subsidies, in 1960-61, poultry was third on the list with £40,000,000, with fatstock £46,000,000 and cereals £52,9000,000. Government economists must therefore realise that poultry and poultry produce were important. It was more or less accepted that, at the moment, the number two animal on the farm in Britain was the hen.

The first speaker to explain disease diagnosis was Dr. W. P. Blount who was very well known and needed no introduction. Obviously, once the disease was there one must be able to diagnose it.

The second side was control, which was going to become more and more important. The emphasis of the whole Congress had been on preventive medicine. It would be seen that the poultry industry was a modern industry. It was much more modern than the dairy industry, or sheep or fatstock. Preventive medicine was a "must." An outbreak of disease meant that the profit was gone; the profit margins were not very great. Intensivism was an absolute wonderland for the spread of infectious disease, so the whole approach had to be prevention, and on that particular subject, Mr. J. D. Blaxland, whom they all knew both from published scientific papers and previous Congresses, would speak. The discussion would be opened by Mr. P. J. Dalton who was one who had "seen the light"; there was an economic return in poultry practice he was principal of a practice in the West Country and had said before the meeting that no other person in his practice would take an interest in poultry to which he (the Chairman) replied, "Don't put it that way: put it that you were the one to see the future in it." Mr. Dalton was spending capital on a diagnostic laboratory within his practice and was, himself, specialising in poultry. Those 3 gentlemen were in front of the audience and he hoped that, in the discussion, members would not only ask questions but also give opinions.

Dr. W. P. Blount, introducing his paper, said that, in preparing it, he had started with symptoms and would have gone on with *post-mortem* findings and field data, but the symptomology field was so large that he had thought it would be enough for one afternoon. The paper was presented in the form of apologies to those who had hoped for more data in the paper, and also for the fact that he had got no voice recordings, which he had hoped to bring.

He recommended as an excellent book, the "Laboratory Handbook on Diagnosis of Poultry Diseases," by Dr. H. P. Chu. The recognition chart in his paper was only the beginnings of what should eventually be a satisfactory chart; it was a template on which one could build a better story. *Post-mortem* results would be worked into the picture and linked with field data, after which there should be quite a reasonable story to tell concerning poultry and diseases. In the chart, he had not mentioned the faeces and the many diagnoses which could be linked with the state of the faeces as such, which indicated the many deficiencies there were in the paper.

Dr. Blount then showed a number of slides illustrating various poultry diseases.

The Chairman thanked Dr. Blount. He said that he had recently had the opportunity of visiting the United States for 6 weeks and, from California to Connecticut, people referred to one particular book which was the one Dr. Blount had mentioned; they said that it was the best text-book that had come from this side of the Atlantic—it was the only book.

The diagnostic side had been emphasised and several conditions shown which were of little significance from an economic angle, in that the birds were still profitable and still laying. He had also shown a number of normal birds; he himself found it embarrassing when certain professional colleagues looked at normal birds and classed them as diseased. It was important to know the normal animal before one started delving into the abnormal.

The next subject was one of major importance in the industry to-day. Intensivism was coming; it was present. The profession had to play its part alongside any intensive diseases and had to do its utmost to bring about preventive programmes. That had to be done in a practical way so that the farmer could appreciate and follow. He hoped Mr. Blaxland would emphasise the control side.

Mr. J. D. Blaxland gave a brief summary of his paper for the benefit of those who might not have had the opportunity to read it before the meeting. He also included reference to some new preparations about which information had come to hand since his paper had been prepared.

The Opener

The Chairman thanked Mr. Blaxland for his introductory remarks. He said that the farmers' "hands had been held" since 1939 with many millions of pounds in subsidy. He did not know the total cost of the fowl pest eradication policy. The broiler industry apparently could pay its own way. He did not know how many people read the "Financial Times" but they could look at poultry industry values from that angle and was very much in favour of that industry doing something for itself; he noticed that the next speaker was far-sighted enough to start poultry specialisation within a general practice. He made a plea that there should not be any more laws; there were sufficient laws in the country, and every time extra legislation was evolved it was adding to taxation. The industry could stand on its own feet, and he thought the European Common Market would be useful in maintaining this. He then called on Mr. Dalton to open the discussion.

Mr. P. J. Dalton, opening the discussion, said that it was with the greatest trepidation that he agreed to open the discussion on papers presented by colleagues who, in their respective fields were recognised authorities.

His own so-called laboratory did not cost £20,000, nor £10,000; it cost about £60, but they were in the process of building a new one because the industry were being kind enough to pay for it, although they did not realise it at the moment.

His interest in poultry was first started because he got a bee in his bonnet about A.I., which the Swiss called Eidelweiss. He rather felt that when A.I. was first brought to this country, people of his age, with one exception, sat on their backsides and did nothing about it in the profession. There was one A.I. centre which was controlled by a veterinary surgeon and it was the best in the country; the rest of the people of his age did nothing about it. He had been in poultry work for a couple of months when he suddenly realised he could change his Ford Anglia for a Prefect, if he kept it up long enough; he was now driving a Zephyr!

He thanked the Organising Committee for asking him to open the discussion. In the two papers, there was material which would be invaluable to the practitioner who was interested in that particular sphere of veterinary science. He was sure that the audience would wish him to express on their behalf sincere thanks to Dr. Blount and Mr. Blaxland for having given the benefit of their years of experience.

They had made a valiant effort to cover a vast field in a short space of time, and it lay with the audience to try to help them fill in the gaps—which were few—by asking questions during the discussion.

Perhaps he had been asked to open the discussion because, being a general practitioner, he might be able to raise the level of the discussion to that of practitioners, and he hoped they would be spared any excess tub thumping by the experts, especially in the field of dietetics and genetics.

Often, he had found it extremely difficult to obtain straight answers to practical problems, and it would appear that very little field investigation had been carried out into those problems, or at least, if they had, the findings had not been published, and as there were present in the audience and on the platform, colleagues who must have a wealth of information available, now was surely the time to try to winkle that information out of them.

Some of the points on which he personally would like information were:—

What was the importance of ascarid, capillaria and heterakis infestations in replacement and laying flocks and in broiler flocks? There was a lot of economic value in the laying flocks, and one could not have a broiler unless one had an egg; one had to have a hen to produce that egg.

Dealing with intestinal complaints, might they have some expression of opinion on the vast problem of coccidiosis? In his laboratory, they were continually running into cases which failed to respond to recognised methods of treatment and that was presumably due to developing resistance or to mixed infection.

From their observations, it would appear that, at present, 4 main species of *Coccidia* had to be contended with, and often mixed infection was found. That made it essential that a diagnosis, not only of the disease but also the species implicated, be made before treatment was instituted. Was it too much to ask laboratories to amplify their reports of "intestinal coccidiosis" to include the species causing the trouble?

The effect of coccidiosis in egg production in layers could be severe, but the treatment might be even more severe. He still shuddered when he recalled the time he prescribed sulphadimidine at the level recommended on the pamphlet enclosed with the bottle, for a flock of 1,000 laying birds which was producing at a level of about 60 per cent. but was losing about 5 birds a week with a mixed *acervuline/maxima* infection. The losses stopped but egg production dropped to 10 per cent. and never really recovered. He had never been asked to go back!

He had been advised that, for the following species of *Coccidia* the drugs of choice were:—

E. Maxima and acervulina: Sulphamezathine and Nicarbazin were to be used for treatment and Amprolium for prevention.

E. necatrix: nitrofurazone for treatment and Amprolium or Zoalene for prevention.

E. tenella: Sulphaquinoxaline and Sulphamezathine for treatment and Amprolium for prevention.

Several questions arose from that. One could not administer Nicarbazin to layers and Sulphamezathine to depressed production. How did one treat *maxima* and *acervulina* infection in layers so as to minimise depression in production?

Certain strains of *tenella* were emerging which were resistant to the sulpha drugs: what could be done? Would the authors agree with the list of drugs for coccidial species he had quoted? They had been told that a combination

of two sulphonamides having a synergistic effect might be the answer.

A lot of commercial poultry suppliers did not use coccidiostats in the rearing stage in order that their birds might pick up some immunity, but it was unlikely that they would be exposed in the rearing stage to all 4 species. Might it not be better to use a coccidiostat and then try to formulate a plan for birds to be exposed in a controlled manner when they finally reached the premises on which they were to spend their adult life, perhaps by means of the intermittent administration of a coccidiostat whilst the oocyst content of the litter built up gradually? There was one more example of where the veterinarian on the spot could well advise, but he would have to acquaint himself with the past history of the farm, and he assured those present that the modern poultry-keeper was only too willing to pay for the services of an informed veterinarian to assist him in planning a programme which would prevent disease; surely that was what must be aimed for in all fields of veterinary science. Here was one field ready tilled.

While discussing coccidiostats, mention should be made of the dangers of starting treatment, especially with sulpha drugs, before an accurate diagnosis had been obtained. He was convinced that there was a condition which was loosely termed non-specific enteritis, which when it affected broiler flocks, could give a picture very similar to intestinal coccidiosis, and he felt that some of the outbreaks of haemorrhagic disease which occurred following the administration of sulpha drugs might well be due to the effect of medication on that condition.

While dealing with parasites, what about ectoparasites? It could be easily decided which of the various preparations were most potent against the different types of infestation, but could they have advice about the practical method of application? To handle each individual of a 5,000 bird flock and give a squirt with an aerosol up the vent was not amusing to the chap who had to do it. Could one use a microsol or smoke generator with birds in the house? Would that penetrate the feathers to control fleas, lice and Northern mite, which did not leave the bird? Was it any use incorporating insecticides in the litter and nest boxes, and which of them could be used without danger of egg taint?

There seemed to be little doubt that there was considerable difference in the pattern of various diseases under various conditions of management, especially when respiratory diseases were considered.

Could anyone tell him what was meant by "a cold" in poultry? Was it a PPLO infection? Was he being too optimistic to hope that it might be possible to differentiate colds, coryza, I.L.T., L.B., and was he being even more optimistic to hope that, in the near future, there might be sufficient laboratories available which could differentiate those conditions for the practitioner? If he was maligning the services already available, could anyone tell him where he could send material from those conditions?

It might be well to mention here the importance of examining birds in the early stages of any disease, especially respiratory diseases, because by the time secondary infection had set in, the symptoms and lesions of the primary condition might well be obscured.

When one considered that the poultry pathologist's bible, Bicester and Schwarte, ran to some 1,200 pages, the authors were to be complimented in covering as much ground as they had in the time and space available to them, and he felt they would excuse him if he mentioned one or two conditions which had interested him in Wiltshire and which were not mentioned in the papers.

There was no doubt in his mind that the condition reported by Sevoian, infectious avian hepatitis, was widespread in this country, and it had been possible to recover a *Vibrio* in certain cases which was considered to be typical of the condition described by Sevoian, although, with the limited laboratory facilities available, it had not been possible to investigate the condition as fully as was intended when better equipment was available. He was certain that that condition was often misdiagnosed as pullet disease, and for that reason, they advised the use of furazolidone for both conditions, although recently, someone had queried the effect of that drug on hatchability.

Dr. Blount mentioned breast blisters in broilers. Some years ago, they had started looking into it to see if respiratory rate and body conformation were in some way implicated in the condition. Birds had been collected from a packing plant; they were of equal weight from the same flock, but some had blisters and some had not. X-rays were taken of the carcases. When the plates were superimposed, it appeared that the angle of the breast bone of the birds with blisters differed from that of unblistered birds. It appeared also that length of leg might also have something to do with the incidence of the condition.

Unfortunately, the packing plant which first asked them to look into it were unwilling to pay for the work which would have had to be carried out in order to reach any conclusion, so it had to be dropped.

They noticed the relatively high incidence of torticollis in turkeys affected with infection of the air sacs, and held the theory that that was due to infection of the middle ear by causal PP.O. Would the authors agree?

Torticollis brought him to the 64,000 dollar question and he hoped that the whole discussion time would not be taken up with the answer. Mr. Blaxland stated that the slaughter policy was "equally successful" in controlling the less acute type of Newcastle disease. Personally, he could not see how anyone could make such a statement when, each week in THE VETERINARY RECORD, there were long lists of affected premises, and one heard that £10,000,000 or thereabouts was paid out yearly in compensation.

He appreciated that authorities such as the speakers had to be a little cagey about what they wrote, but he asked them point-blank for their personal—and he stressed "personal" not official—views on the method presently in force to try to cope with fowl pest. He himself was convinced that they would have to resort to vaccination.

In general practice, poultry work was a field of veterinary science which was crying out for help; it was a fascinating field in which practitioners could exercise their training, knowledge and gifts to the full: an opportunity not to be met in any other aspect of their work. The economics and practice of poultry-keeping nowadays were such that there was tremendous personal satisfaction and financial rewards for the profession, and unless practitioners were prepared to equip themselves in that work, a vacuum would be formed—in fact it had been formed. And into that vacuum would be sucked non-veterinary advisers who would take the place which could, and should, be occupied by the qualified veterinary surgeon. If those in the profession did not get cracking now, it would be no use belly-aching later!

The General Discussion

Mr. Blaxland said that he had listened to Mr. Dalton's comments and observations with considerable interest; it was always helpful to have the opinion of a colleague working in the field concerning the recommendations and advice put out from the laboratory for the control of the disease.

Worm infestations in his experience were not of great importance to-day. Occasionally heavy infestations were encountered, interfering with egg production, but such outbreaks were comparatively rare and frequently trouble had in fact arisen in the flock usually from a combination of factors. Worm infestations appeared to build up in individual debilitated birds, particularly following a check or setback through adverse environments or inter-current diseases. Flock medication was seldom justified and he was inclined to regard most of those cases diagnosed in the laboratory *post-mortem* room as being individual culled birds. If treatment was necessary, however, he could only refer Mr. Dalton to the preparations listed in the table contained in the paper.

On coccidiosis he wished to stress that the emphasis should always be on prevention rather than actual treatment of an outbreak. The aim should be the establishment

of resistant flocks through constant exposure during early life to non-pathogenic levels of infection. On encountering an outbreak of coccidiosis in the field, the first consideration should be—not which drug and at what level it should be administered, but why should it be necessary to resort to treatment at all on this particular farm? Why are there still apparently a large number of susceptible individuals in this flock? If coccidiosis was approached in this manner a veterinary surgeon's client would obtain lasting benefit. There were a number of reasons why outbreaks of coccidiosis still occurred under some field conditions, in spite of the use of coccidiostatic drugs in the food. It could happen that the ground on which young birds had been reared had been too clean so that the level of coccidial infection present was insufficient to stimulate a lasting immunity; under such conditions fowls might remain fully susceptible to infection, an outbreak of coccidiosis occurring when eventually the flock was moved to its laying quarters, probably a deep litter house in which the level of coccidial infections was likely to build up rapidly. Furthermore, it was known that certain of the coccidiostatic drugs when incorporated into rearing mashes proved, in a sense, too effective in that they interfered with the development of immunity. This occurrence might be prevented by periodic intervals during rearing without a coccidiostat in the food. Finally, Mr. Blaxland advised against the treatment of a flock of adult fowls merely on the confirmation of coccidiosis in one or two individuals without any evidence of progressive disease in the flock as a whole.

Mr. Dalton had asked if laboratories when giving a diagnosis of coccidiosis could not specify the species of *Coccidia* involved. In the Scientific Exhibition staged by the Congress authorities, there was a demonstration on this very point. It was possible in the diagnostic laboratory to suggest the probable coccidial parasites present, based on the distribution of the lesions produced; in practice however, many outbreaks proved to be due to mixed infections and it was not possible within the time available for the carrying out of a routine examination to identify accurately all the species, other than to distinguish between the intestinal and caecal forms of the disease. At Weybridge they did not think that an accurate diagnosis of coccidial species was always strictly necessary or that it made much difference when it came to the advice to be given by a veterinary surgeon. In their view, the sulphonamides and the new drug, Whitsyn S, were the most efficient preparations available for the actual treatment of a clinical outbreak of this disease, no matter which coccidial species was involved. The real problem was to decide whether or not to resort to flock medication, and this was something more easily settled by the man on the farm with the field picture before him rather than by his colleague in the laboratory.

Mr. Dalton had mentioned the difficulties of treating birds individually in outbreaks of ectoparasite infections. At Weybridge they had not had much practical experience of the treatment of these conditions; on most premises parasites appear to have been controllable by use of the insecticidal sprays and aerosols available on the market. Occasionally very heavy infestations of northern fowl mite were encountered, when it became necessary to treat the affected birds individually, treatment being required on more than one occasion in order to overcome the infection.

Respiratory diseases of poultry were, of course, a major problem, and their diagnosis and treatment presented many difficulties to both the laboratory worker and veterinarian in the field. An accurate diagnosis based on the isolation of the causal organism might take many weeks to complete, and from the practical point of view the result of laboratory tests and examinations made little difference to the measures it was possible to take for the treatment of these diseases at the present time. Antibiotics, either by injection or in the food, or water, were beneficial in some outbreaks, but the important part played by adverse environmental conditions as predisposing factors in the causation of these diseases should never be overlooked. There were plans at Weybridge for a more detailed study of these diseases, and it was to be hoped that this might lead to a greater understanding of their prevention and control. At the present time they could not undertake routine investigation of field outbreaks for veterinary surgeons in practice.

On the problem of Newcastle disease control, Mr. Blaxland said that he was prepared to give his personal opinion, but he wished to emphasise that his views must not on any account be taken as representing those of the Ministry, or as an indication of future policy.

This was a problem on which the profession should be well informed and he had always felt that it was important for the veterinary surgeon who wished to be accepted by his poultry keeping clients to appreciate some of the current disease problems of the poultry industry. It was expected that the report of the Independent Committee appointed by the Minister to review the fowl pest policy would be published towards the end of the year, and the poultry farmer would certainly expect his veterinary adviser to be able to discuss the recommendations with him.

Mr. Blaxland thought that too many dogmatic assertions were made on the control of Newcastle disease by individuals with insufficient evidence and experience at their disposal. It was his view that a disease problem should be examined from all possible angles before firm views were expressed for or against a particular policy. In criticising a policy some of the points to be considered were the nature of the disease involved and its impact on the industry and the experience of other countries in controlling it; the estimated cost of the policy, first in public money from the Treasury, and secondly the cost to the industry of the control measures; the possible effects of the policy on the incidence of other diseases; the manpower available for carrying out the policy, first on the staff of a government service, and secondly, availabe on farms to carry out operations, such as vaccination.

In support of the present policy it could be claimed that the acute form of Newcastle disease introduced into the country in 1947 had been eradicated and only 2 or 3 years were required to do so. It could, furthermore, be claimed that the present policy did, in fact, control the mild form of the disease; despite the fact that serious epidemics were not infrequent they had, so far, always been brought under control within a few months, and for long periods during the last 10 years or so, only a small number of outbreaks had occurred. The disease had been virtually eradicated from Scotland, Wales, and most of the South-West of England. It could not be denied, however, that the administrative costs of this policy, running into millions of pounds, was one of the major factors leading to the appointment of the Independent Committee. Nevertheless, it should not be forgotten that the Newcastle disease position in this country, by virtue of the present policy, was probably better than in many other parts of the world.

The unrestricted use of live vaccines for the control of Newcastle disease was not the answer to the problem, and despite the opinions expressed in many quarters he did not think it could be claimed that vaccination was really the complete answer to the problem even in the United States. In support of the that view, he quoted from a paper presented at the 64th Annual Meeting of the American Veterinary Medical Assocation in New York last year, which emphasised the need for a re-evaluation of the Newcastle disease programme: a paper presented by prominent veterinary authorities in the United States, the one country which was often held up to us as the model that we should follow in controlling Newcastle disease. The authors of this paper claimed that effective control of Newcastle disease had *not* been accomplished by using live virus vaccines. The disadvantage of the live vaccines was that they perpetuated the disease and might even produce it if not properly used. It was pointed out in the article that vaccines were stress agents which precipitated latent infections of other diseases, particularly air sac infection and other respiratory diseases. It was further claimed that the increase in condemnation of poultry carcases in the United States was due largely to down grading and rejection of carcases because of the presence of respiratory disease. Research had shown that air sac

infections were precipitated by the use of live virus vaccines. The paper ended by urging that there should be consideration of the control of Newcastle disease through the use of a vaccine which would not stimulate the disease nor precipitate latent infections. Mr. Blaxland explained that he had quoted this paper to show that there was no generally accepted satisfactory way of controlling Newcastle disease at the present time.

There had in recent years been encouraging results following the use under laboratory conditions of Newcastle disease dead vaccines. Adequate immunity was claimed following their use, and there was no danger of precipitating outbreaks of other infections. There was, furthermore, no risk of disseminating egg transmitted diseases, a real danger, even under the most stringent controlled conditions when the virus strain itself had to be propagated on embryonated chicken eggs. One of the big difficulties, however, with a dead vaccine was that each bird had to be injected individually; Mr. Blaxland, however, wondered whether even this difficulty was insuperable, and whether ways and means might not be found of handling broiler flocks satisfactorily in order to permit vaccination with a dead vaccine; further research might even lead to the discovery of other means by which dead vaccines could be administered without loss of effect. The possible use of dead vaccines in the control of Newcastle disease in this country should, in his opinion, be considered more seriously before the irrevocable step of introducing live virus vaccines was taken.

The Chairman said that an egg production graph had been shown on the screen of pullets coming into lay and a dip in the production curve had been shown after an attack of Newcastle disease, with a subsequent rise; the rise had attained the point where the egg production would have been had they not had Nawcastle disease, also the point had been made that the birds had had no treatment.

On the recent Newcastle Disease Commission it depended who one visited in the United States as to what the opinion was. All he could say was, how did one assess the efficiency of an industry? By using live vaccine such as they had in the United States that was "supposedly" detrimental, their industry must be inefficient but it was known that the Americans exported to this country, not officially but unofficially, and wherever one went in Europe one saw United States poultry, so whether, in the eyes of their own Government or their own Government veterinarians, it had been a step in the wrong direction, he did not know. However, the efficiency of their industry was such that it had currently shown that it could, periodically, make our broiler industry shiver, in that they could threaten Britain with massive imports of cheap broilers "oven ready."

From the breeding stock angle, both this country and other European countries had accepted American stock and breeding methods. They had a full egg market themselves. In 1959, each person ate 313 eggs as opposed to 240 in this country. Each person in America ate 34.8 lb. per year per head of broiler meat as opposed to 11.4 lb. per head in the U.K. Taking the over-all picture, did one assess the efficiency of an industry through the eyes of the "cautious veterinarian" or did one take notice of economics and from the point of view that here there was a flourishing industry satisfying its home market and able to export?

Dr. W. R. Wooldridge (The Animal Health Trust) said that, in view of the remarks on Newcastle disease, it might be as well for him, as Chairman of the Committee of the B.V.A. that considered the matter, to state quite categorically that the B.V.A. had suggested that the policy should be changed. One of the arguments in favour of not changing it was that the respiratory diseases were encouraged to spread throughout the nation's flocks if vaccine was being used. Although the vaccine was not used in this country the growth of the respiratory diseases in hatcheries and the broiler industry was staggering and hence that argument for not going in for vaccine had been weakened.

The Committee, that went into the matter carefully, had proposed a change. They had not proposed a complete change but a modification whereby the slaughter policy should be retained should an acute form of the disease arise.

The question was raised also on the use of the fowl typhoid vaccine. The amount in use in this country had been growing enormously, with considerably more use in the second year, so presumably good results were obtained. There had been practically no complaints with regard to its efficacy. Although it had not yet been published, he could say that it had now been possible to produce a dried vaccine which would make storage easier and also be available for export.

It was most encouraging to see from the enormous attendance at the meeting that everyone was taking such an interest in poultry disease. He congratulated Mr. Dalton as a practitioner for going ahead in the way he had.

Mr. W. H. Parker (Thirsk) wished to raise a point under the causes of losses in young poultry not mentioned at all, which he considered to be by far the biggest cause of loss. That was the consumption of fibre. All the specific diseases that had been referred to might be important, but in his experience, none of them caused anything like the amount of loss that fibre in the digestive system was responsible for.

Usually, in really young birds, it was the picking up of bedding; turkeys were particularly prone to that, and in the West Midlands, turkey rearers were sufficiently educated to rear their birds on bare boards without any fibre for them to pick up. Up to the moment, he had found that such an idea was quite unacceptable to the North of England but recent change of personnel might lead to enlightenment.

With older birds, there was consumption of coarse grass which made a complete mat and resulted in death. He considered that the biggest single cause of death of birds up to the age of about 3 months. The reason for their perverted appetite was not always easy to find, but it was very important, if one was going to a farm to advise, that different forms of poultry management were understood, because it was essentially a management problem. Very often, it might be primarily a problem of trough space. It was not only the amount of troughs available, which many farmer kept below the necessary amount, forgetting that birds grew rapidly, but birds ate and drink alternately and troughs had to be reasonably near the drinking water or the birds could not be bothered to go back to the troughs.

He had a suspicion that the appetite was liable to be depraved following the use of sulphonamides, which was a difficult problem to cope with if coccidiosis was present. On the other hand, more people used sulphonamides at the slightest sign of ill-health without any evidence of this disease.

Dr. Blount said that he was certain it was responsible for some of the non-specific enteritis mentioned by Mr. Dalton. Amongst other things, it was due to the fact that birds were naturally inquisitive and wanted to peck downwards. They were standing on wood shavings and if there was a shortage of trough space or if they were not particularly happy and got chilled, they would peck the litter. It would pay if veterinary surgeons opened every gizzard because they would find a large amount of wood shavings in many of them. He hoped that those who reported on *post-mortem* examinations would pay as much attention to that as to other features, because it might take away the mind of the farmer from the food to the litter.

In the case of turkey poults, when they were bedded on chopped straw or wood shavings, many farmers put down 2 in. to begin with and, when 6 weeks old, put down a new lot. The new lot being new and clean, the birds pecked it. An important aspect of poultry husbandry had been brought out.

Mr. L. A. Hemsley (Buxted) commented that this was the first time he had heard what the B.V.A. had told the Plant Committee. He was sorry to hear from Dr. Wooldridge that the Association apparently had advocated the use of live Newcastle disease vaccines, and that they had reiected the objection that live vaccines would spread other diseases on the grounds that such diseases were already widely

spread. It was difficult to control respiratory disease when the causes of it were not known. At present it was not possible to obtain help in the diagnosis of respiratory disease. Although he agreed with Mr. Blaxland that there was little point in identifying the actual cause of an individual outbreak of respiratory disease, it was important that we should know what were the main causes of respiratory disease in the nation's poultry flocks.

Although he agreed that live Newcastle disease vaccines might be quite successful in protecting young stock intended for egg production, he pointed out that such birds had several months to recover from the effects of a live vaccine before coming into lay. This was not true for broilers which were vaccinated as they were reaching their period of maximum production. He could not see Dr. Wooldridge's point in regard to respiratory disease having been spread already. Surely something should be done to enable that spread to be controlled, such as the establishment of a unit for the proper diagnosis of respiratory disease? Was it necessary to make things worse by adding Newcastle disease to those agents already present? At the moment losses due to respiratory disease in the average broiler unit in this country were relatively low. He emphasised that even so it was the most important disease group of all, indeed the other diseases might almost be ignored for they were hardly of any importance compared with respiratory disease. At present it was not disastrous for the average broiler unit to get respiratory disease; but if Newcastle disease was added losses could approach those experienced in America, which might amount to over half the birds in a group.

The Chairman said that the question was being asked why the Newcastle disease problem was still subsidised by the taxpayer. There was an assumption that 100 per cent. of poultry in the country would be vaccinated against Newcastle disease. His company had about 750,000 birds spread throughout Great Britain and Eire. They would not necessarily use Newcastle disease vaccine in every flock. Hygiene was a factor that could play its part in prevention. There was a lot of genetic background to breeding of the broiler but this bird had not been bred with any particular emphasis on disease resistance. It had been bred with two ideas in mind; food conversion and rapid weight gain and as low a sex differential as could be obtained. With the egg production chicken which had been selectively bred since 1926, those things had been included.

Mr. W. W. C. Gibson referred to the excellent summary of drugs available for the treatment of poultry diseases contained in Mr. Blaxland's paper, and pointed out that where Amprolium was mentioned, it was shown as being used only at 0.0125 per cent. In some cases lower levels were being used for replacement birds. He mentioned this in case some practitioners who came across it might feel the drug was not being used correctly.

Mr. Blaxland had paid particular attention to the question of co-operation with feeding-stuff firms where the quality of the food was suspect. He pointed out that quite often, where analysis of food was made, it was extremely difficult, on an initial analysis, to find anything wrong with the food. It was also often very difficult to reproduce the condition by test feeding of suspect food. Dr. Blount might support these points from his longer experience with that type of difficulty.

Dr. Blount, in his paper, mentioned some points on nutrition. Could he amplify a little so that practitioners who have analyses of the food carried out would be better placed to interpret the results?

Dr. Blount stated in his paper that "a fall in production from 38 to 50 per cent. has been recorded following the use of rations containing too little phosphorus." Should this read 83 to 50 per cent. and what did "too little phosphorus" mean?

Dr. Blount also said that 2 to 5 per cent. of cracked eggs was normal, but when it increased to 7 to 10 per cent., the possible causes included an excess of calcium. He thought that was not correct because, in view of the higher calcium levels now being incorporated in some of the commercial rations, anything up to 4 to 5 per cent. should be taken as a normal calcium level for laying birds.

Finally, he said that he was certainly aware of the difficulties in the relationship between the veterinary surgeon in practice and the representatives of commercial firms in the investigation of poultry diseases. The situation, which was a difficult one, can only be resolved if approached in a spirit of co-operation on both sides rather than with a feeling of animosity.

Dr. Blount said that the fall in production was for 2 groups of birds; one went up 30 per cent. and the other 56. The paper did not give levels of phosphorus but it was known that no more than 0.45 per cent. was required, so it was presumably about 0.3 per cent. of less.

On the question of cracked eggs and excess calcium, he was not sure but he thought it could be assumed that because 4.75 per cent. of calcium could normally be given with good results, it was obviously in excess of that. In practice, it meant that the farmer was feeding a ration containing some calcium and instead of giving the birds a small amount, put in front of the birds a large quantity; some birds would then take in excess of normal.

He asked Mr. Dalton to amplify the question of infectious avian hepatitis; he had not met it. Could he say more about species, age, symptoms, *post-mortem* features, etc.

Mr. Dalton said that he had some slides. The disease affected poultry rarely under 10 weeks of age. It caused a spotty mortality and was due to a *Vibrio*. He had doubts about the technical work of Sevoian; he thought he was sound. Apparently, the *Vibrio* could be isolated. There was blood agar from 25 per cent. to 100 per cent. of yolks at inoculation of a 6-day egg.

Post-mortem lesions could be rather vague; they could look like pullet disease. It depended on the age at which a bird was affected. Losses were seldom over 5 per cent. There was muscle degeneration in the heart and small necrotic areas of varying amounts in the liver. There was usually no nephritis as in pullets disease.

Mr. Dalton then showed slides to illustrate his remarks.

The Chairman said that it was plain from the amount of controversy among the experts that there was much more research required and he asked them to take from Weybridge Laboratory and Veterinary Investigation Centres as much of the routine *post-mortem* work as possible. Mr. "X" had coccidiosis last year, he has it this year and will have it next year; it was such examinations which, undoubtedly, held up the laboratories and hampered research work. Therefore, if some practitioners had been stimulated to take an interest and start work on poultry disease, it would undoubtedly help the research institutes.

On behalf of those present he thanked Dr. Blount, Mr. Blaxland and Mr. Dalton. He thanked the audience for listening and for making the afternoon so lively.

THE REGISTER OF VETERINARY SURGEONS

The undermentioned were admitted to membership of the Royal College of Veterinary Surgeons on December 16th, 1961, in virtue of the registrable degree of B.A., Vet.M.B., of the University of Cambridge: Brown, Keith Michael Treves, "Rivelin," Hillside Road, Pinner Hill, Middlesex; Rozenblum, Myriam (Miss), 132, Dollis Hill Lane, London, N.W.2.

M. PAUL GODECHOUX

We record with regret the death of M. Paul Etienne Godéchoux, a former President of the French Veterinary Association. He died at the age of 75 on December 6th in Paris. His wife is well known for helping to arrange exchange holidays with the children of French veterinary surgeons, and is President of the Association Francaise des Femmes de Veterinaires.

News and Comment

EXTRAORDINARY GENERAL MEETING OF THE BRITISH VETERINARY ASSOCIATION

7, Mansfield Street,
London, W.C.1.

Notice is hereby given that an Extraordinary General Meeting of Members of the British Veterinary Association will be held at the Royal Society of Medicine, 1, Wimpole Street, London, W.1, on Friday, January 19th, 1962, at 2.15 p.m.

To consider, and if thought fit, confirm the unanimous recommendation of the Council of the Association:

THAT the Articles of Association shall no longer apply to this company and that in lieu thereof the new articles of Association already prepared, a copy whereof has been initialled by the Secretary for the purpose of identification be adopted by the company.

JOHN A. ANDERSON,
General Secretary.

December 19th, 1961.

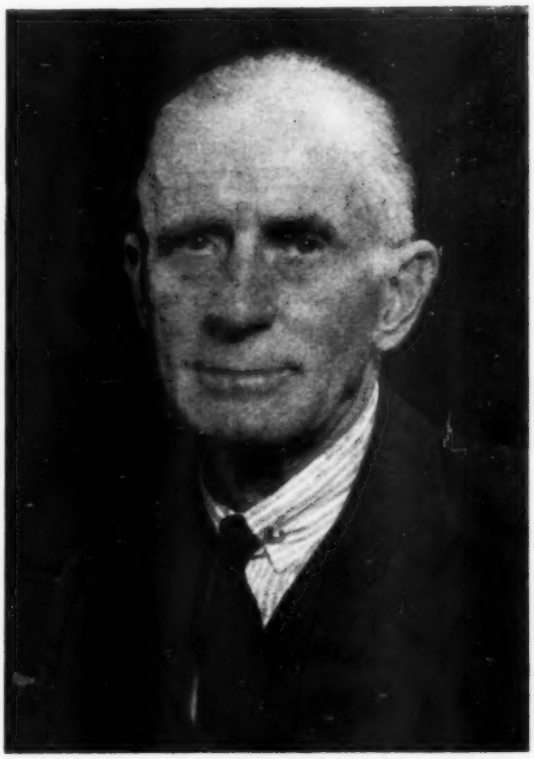
NOTE: *A copy of the new Articles of Association referred to in item of the Notice can be inspected by any member at the offices of the Association, 7, Mansfield Street, W.1, on any week-day (except Saturday) between the hours of 9.30 a.m. and 5.0 p.m. without fee.*

DR. H. H. GREEN, O.B.E.

We are indebted to two senior workers at Weybridge for the following memoir.

His many friends and colleagues in this country and abroad will have noted with regret that H. H. Green died on December 4th, 1961. His life's work was concerned with the application of biochemistry to the elucidation of problems of health and disease in farm livestock.

Born in South Africa of Scottish parentage in 1885 he had a distinguished academic career at the University of Glasgow where he graduated in chemistry with physiology, geology, mathematics and physics, and attended courses at the West of Scotland Agricultural College, and the Royal Technical College, Glasgow. During 1912-14, following the award of an 1851 Exhibition Scholarship which at that time was regarded as a high scholastic achievement, he proceeded to selected Ph.D. courses at the University of Leipzig in agriculture, animal husbandry and bacteriology, and in 1920 studied physiology and biochemistry at the University of Yale, U.S.A. He obtained his Glasgow D.Sc. in 1916 and was awarded a D.Sc., *hon. causa*, by the University of South Africa in 1935. Green was appointed Assistant to the Professor of Agricultural Chemistry in the West of Scotland College of Agriculture in 1910, and Grieve Lecturer in Physiological Chemistry in the University of Glasgow in 1912.



Following his period in Leipzig, he went to South Africa in 1914 as Biochemist in the Division of Veterinary Research, and in 1920 was appointed Professor of Biochemistry in the University of the Union of South Africa combined with a Sub-Directorship of the Veterinary Research Institute at Onderstepoort. He occupied this combined post involving teaching, research and various administrative duties such as secretaryship of the Veterinary Faculty, and technical advisory work to the Secretary of Agriculture, until a break-down in health necessitated his retiral from the South Africa Service in 1929. After his recovery he held a temporary post at Wye Agricultural College in 1931 and came to Weybridge in 1932 financed by a grant from the Empire Marketing Board. He proceeded to develop the nucleus of a biochemistry unit with the help of two voluntary workers until in 1935 he was appointed Senior Research Officer of the newly-created Biochemistry Department. During the next 3 years 2 junior research posts were added to his department

which subsequently developed rapidly to become one of the largest departments at the Central Veterinary Laboratory. In 1949 he was awarded an O.B.E., and in 1953, in recognition of his services to veterinary science, he was elected an Honorary Associate of the Royal College of Veterinary Surgeons. He retired from his post at Weybridge in 1953.

Following the time of his appointment in 1914 as the first chemist to the Division of Veterinary Research in South Africa, Green occupied an almost unique position in the scientific world. His amazing versatility, his wide knowledge, his quickness at visualising the biochemical implications of any veterinary problem presented to him, won him an unrivalled place in veterinary science: he could truly be designated a pioneer in the field of veterinary biochemistry first in the Union of South Africa where he was remote from other biochemists, and secondly in Great Britain at a time when the biochemical aspects of animal health were not appreciated. His work in South Africa, much of it carried out in collaboration with the late Sir Arnold Theiler, will always be regarded as classical, particularly that on aphosphorosis in ruminants, arsenical dips and poisoning, and deficiency diseases.

In this country, his contributions to the subjects of toxicology, metabolic disorders of ruminants, the major and minor mineral requirements of livestock and the diseases caused by their deficiency, are outstanding. Particularly noteworthy was his development of rapid and accurate micro- or semi-micro-analytical methods for use in large-scale field testing and diagnostic procedures. His name, too, will always be associated with the development of P.P.D. tuberculins for use in the veterinary field. The expansion and range of interests of the present Biochemistry Department at Weybridge are practically entirely attributable to his genius, drive and initiative.

After his appointment to the Chair of Biochemistry in the University of South Africa, Green became an enthusiastic teacher and a member of the senate equally popular with colleagues and students. His liking for teaching and association with younger minds was evident throughout the 20 years he was at Weybridge. Not only did he make great advances in his chosen field but he also gave keen interest and support to projects concerned with the post-graduate teaching, training and career prospects of young veterinarians and others, in the fields of biochemistry, physiology and immunology.

A remarkable memory, a quick and penetrating intellect, and an analytical mind were, perhaps, Green's most outstanding attributes. He was shrewd and skilful in debate and argument, and tenacious in the advocacy of developments and expansions which he considered necessary or desirable. His analytical, constructively critical and interesting assessments of practical and theoretical problems and situations were remarkable and related not only to his specialised scientific background and interest, but also to a wide-ranging field of intellectual, political and social topics.

The broad human sympathies and innate kindliness of "H.H." were well known to all his colleagues and associates. Nevertheless he was a strict disciplinarian and often made exacting demands on his staff which were always justified by his concern for accuracy as well as efficiency and speed in solving the problem in hand. The "old man" had the respect and affection of his whole staff and under his leadership the ever-growing Biochemistry Department was a happy and co-ordinated group.

PERSONAL

Marriage

KINCH—CARTER.—On December 12th, 1961, at St. Benets Church, Cambridge, David A. Kinch, B.V.M.S., M.R.C.V.S., of Ainwick, Northumberland, to Wendy K. Carter, of Mildenhall, Suffolk.

Birth

BERGER.—On November 27th, 1961, to Lorna, wife of Felix Berger, M.R.C.V.S., of Medway, Gt. Horkesley, Essex, a son, Jeremy, brother for Simon.

CAMERON.—On December 9th, 1961, to Helen, wife of R. Scott Cameron, M.R.C.V.S., c/o Parke Davis & Company, Hounslow, Middlesex, a son, Paul, brother for Ralph.

JOHNSON.—On December 17th, at 50, Hempstead Road, Watford, to Dinah (*née* France), and Roger Johnson, B'S.V.SC., M'S.R.C.V.S., a daughter (Victoria Margaret), sister for Charlotte and Hugh.

COMING EVENTS

January

4th (Thurs.). B.S.A.V.A. METROPOLITAN REGION. New Year Dinner Meeting at the Royal Society of Medicine, 1, Wimpole Street, London, W.1, 7 p.m.

10th (Wed.). W.C.V.A. DEVON BRANCH. Clinical Meeting at the Church House Inn, Harberton, Totnes, 7.30 p.m.

11th (Thurs.). CENTRAL VETERINARY SOCIETY. Meeting at the Royal Veterinary College, Camden Town, 6 p.m.

24th (Wed.). LANCASHIRE VETERINARY ASSOCIATION. General meeting at the University of Liverpool, 2.30 p.m.

February

2nd (Fri.). ROYAL COUNTIES VETERINARY ASSOCIATION. Annual Dinner Dance at the White Hart Hotel, Sonning-on-Thames, 7.30 p.m.

14th (Wed.). BRITISH EQUINE VETERINARY ASSOCIATION. First meeting at the Royal Station Hotel, York, 9.30 a.m.

SOCIETY FOR THE STUDY OF ANIMAL BREEDING. Meeting at the Royal Society of Medicine, 1, Wimpole Street, London, W.1.

JANUARY MEETINGS OF B.V.A. STANDING COMMITTEES AND COUNCIL

Tuesday, January 16th, 1962—7, Mansfield Street

5 p.m. Finance sub-committee.

Wednesday, January 17th, 1962—7, Mansfield Street

10 a.m. Veterinary State Medicine Committee.

11.30 a.m. Small Animals Committee.

2.15 p.m. Home Appointments Committee.

3.45 p.m. Parliamentary and Public Relations Committee.

Thursday, January 18th, 1962—7, Mansfield Street

10 a.m. Farm Livestock Committee.

2.15 p.m. General Purposes and Finance Committee.

Friday, January 19th, 1962

10 a.m. Meeting of Council at the Royal Society of Medicine: 1, Wimpole Street, London, W.1.

Correction

In our recent note on the British Veterinary Zoological Society, the honorary treasurer/secretary's address was described as Dunstable, Herts; it is of course Dunstable, *Beds.*

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the date on which disease has been confirmed, followed by the postal address and local authority.

Anthrax

December 18th 1961. Mrs. E. L. E. Warren, The Hyde, Handcross, Haywards Heath, Sussex (Parish: Lower Beeding). (Cattle.) WEST SUSSEX.

December 18th, 1961. Mrs. E. A. Durston, Cornish Farm, Shoreditch, Taunton, Somerset (Parish: Pitminster). (Cattle.) SOMERSET.

December 18th, 1961. C. Bailey, Charminster Farm, Wanchurch Lane, Charminster, Dorchester, Dorset (Parish: Charminster). (Cattle.) DORSET.

December 18th, 1961. C. & N. Railton, Holly Farm, Buckton, Bridlington, Yorks. (Parish: Bemton). (Cattle.) YORK, EAST RIDING.

December 19th, 1961. D. W. F. Williams, Walcombe Farm, Wells, Somerset (Parish: St. Cuthbert Out). (Cattle.) SOMERSET.

Fowl Pest

December 15th, 1961. Mrs. A. Frith, 41, Coronation Avenue, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 15th, 1961. E. H. Stocke, Hemsworth Farm, Huggate, York. YORK, EAST RIDING.

December 15th, 1961. G. Andrew, 31, High Causeway, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 15th, 1961. F. L. Newman, Ash Lane, Carleton Rode, Norwich, Norfolk, NOR 03X. NORFOLK.

December 15th, 1961. W. H. Chamberlain, Crown Drove Farm, Christchurch, Wisbech, Cambs. ISLE OF ELY.

December 15th, 1961. W. Dewe, The Wafftons, Surbiton Golf Course, Woodstock Lane, Chessington, Surrey. SURREY.

December 15th, 1961. T. Henson, 15, Queen Street, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 15th, 1961. J. H. Cyster, Sparlands End Farm, Priors Hill, High Wycombe, Bucks. BUCKINGHAMSHIRE.

December 15th, 1961. G. Hailstone, 37, Gracious Street, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 15th, 1961. Mrs. V. Tye, Elms Farm, Carleton Rode, Norwich, Norfolk, NOR 03X. NORFOLK.

December 15th, 1961. Mrs. R. F. Bennett, Cross Green Cottages, Thelmethan, Diss, Norfolk. WEST SUFFOLK.

December 16th, 1961. F. B. Clarke, 7, Roseacre Villas, Pimlico, Kings Langley, Herts. Disease at: Verulam Nurseries, Hartnell Lane, Kings Langley, Herts. HERTFORDSHIRE.

December 16th, 1961. J. E. Lisley, Sladmore Cottage, Cryers Hill, High Wycombe, Bucks. BUCKINGHAMSHIRE.

December 16th, 1961. B. L. Lovell, 78, Eastrea Road, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 16th, 1961. Mrs. K. Fountain, 8, Mactons Close, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 16th, 1961. F. J. Crabbe, Burton Green Farm, Burton, Christchurch, Hants. HAMPSHIRE.

December 17th, 1961. W. T. Aldridge, Long Drove, Cottenham, Cambridge. CAMBRIDGESHIRE.

December 17th, 1961. R. J. Hampden, Garden House, West Harling, Norwich, Norfolk, NOR 15X. NORFOLK.

December 18th, 1961. Mrs. P. V. Searle, 168, Eastrea Road, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 18th, 1961. Mrs. D. A. Cooke, Rose Cottage, Wicklewood, Wymondham, Norfolk. NORFOLK.

December 18th, 1961. N. E. Sparrow, Blackhorse, Styleham, Diss, Norfolk. EAST SUFFOLK.

December 18th, 1961. J. & D. Crabtree, Barkisland Hall Farm, Barkisland, Halifax, Yorks. YORK, WEST RIDING.

December 18th, 1961. C. Wright, 92, Drybread Road, Whittlesey, Peterborough, Northants. Disease at: 9, Cemetery Road, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 18th, 1961. J. A. Bunting, Ewell Hall, Kelvedon, Colchester, Essex. Disease at: Highfields Farm, Kelvedon, Colchester, Essex.

December 18th, 1961. Mrs. R. Witton, Manor Farm, Hopton, Norfolk. WEST SUFFOLK.

December 18th, 1961. W. P. Edlin & Son, Upper Bottom House Farm, Chalfont St. Giles, Bucks. BUCKINGHAMSHIRE.

December 18th, 1961. R. H. Reeve, Church Farm, Thelnetham, Diss, Norfolk. WEST SUFFOLK.

December 18th, 1961. J. S. Lintern Ltd., Hoppers Farm, Great Kingshill, High Wycombe, Bucks. BUCKINGHAMSHIRE.

December 19th, 1961. H. E. P. Mann, The Park House, Tostock, Bury St. Edmunds, Suffolk. Disease at: Green Farm, Tostock, Bury St. Edmunds, Suffolk. WEST SUFFOLK.

December 19th, 1961. A. Mee 10, Delph Street, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 19th, 1961. A. Townley, North Road Bungalow, Reddings, Cheltenham, Glos. BOROUGH OF CHELTENHAM.

December 19th, 1961. J. A. Barnes, 60, Cemetery Road, Whittlesey, Peterborough, Northants. ISLE OF ELY.

December 19th, 1961. J. A. Siddall, Holmlea, Fiddlers Green, Cheltenham, Glos. BOROUGH OF CHELTENHAM.

December 19th, 1961. R. J. Davey, Penrose, The Street, Ashwellthorpe, Norwich, Norfolk, NOR 89W. NORFOLK.

December 19th, 1961. S. W. Green, Cross Lane Farm, Ibsley, Ringwood, Hants. HAMPSHIRE.

December 19th, 1961. K. Nott, Pelham Hall, Twinstead, Sudbury, Suffolk. ESSEX.

December 20th, 1961. C. H. Muskett, Wood Farm, Ashwellthorpe, Norwich, Norfolk, NOR 89W. NORFOLK.

December 20th, 1961. Miss B. Large, West View, Upgate Street, Carleton Rode, Norwich, Norfolk, NOR 03X. NORFOLK.

December 20th, 1961. C. R. Mison, Turnpike Farm, Carleton Rode, Norwich, Norfolk, NOR 03X. NORFOLK.

December 20th, 1961. T. E. Stevenson, Hardicroft Farm, Carleton Rode, Norwich, Norfolk, NOR 03X. NORFOLK.

December 20th, 1961. High Winds Broilers, Bonnywood Farm, Barking, Ipswich, Suffolk. EAST SUFFOLK.

December 20th, 1961. V. M. Clarke, Manor Farm, Wattlefield, Wymondham, Norfolk. NORFOLK.

December 21st, 1961. F. Waters, Privet Lodge, West Harling, Norfolk. NORFOLK.

December 21st, 1961. C. Sweet, Dairy House, Burton, Christchurch, Hants. Disease at: Burton Green Farm, Barton, Christchurch, Hants. HAMPSHIRE.
December 21st, 1961. H. Hemmaway, 8, East Delph, Whittlesey, Peterborough, Northants. ISLE OF ELY.
December 21st, 1961. A. J. Cooper, Deal Tree Farm, Wymondham Road, Bunwell, Norwich, Norfolk. NORFOLK.
December 21st, 1961. J. J. Morgan, Bentley Heath Farm, Barnet, Herts. MIDDLESEX.
December 21st, 1961. B.O.C.M. (Miss E. F. King) B.O.C.M. Poultry Section, Barlby, Selby, Yorks. YORK, EAST RIDING.
December 21st, 1961. H. & E. M. Smithies, Upper Hall Farm, Barisland, Halifax, Yorks. YORK, WEST RIDING.

Swine Fever

December 15th, 1961. A. E. Fisher, Inglewood Farm, Ingoldsthorpe, Kings Lynn, Norfolk. NORFOLK.
December 15th, 1961. G. H. Syson, 11, Claro Avenue, Harrogate, Yorks. Disease at: 102, Dragon Allotments, Claro Road, Harrogate, Yorks. YORK, WEST RIDING.
December 15th, 1961. N. Jones, Kingtor, Church Road, Great Burstead, Billericay, Essex. ESSEX.
December 18th, 1961. C. Watson, Main Street, Willoughby on the Wolds, Loughborough, Leics. NOTTINGHAMSHIRE.
December 18th, 1961. C. A. Davis, Baldwins Wood, Kilcot, Newent, Glos. GLOUCESTERSHIRE.
December 18th, 1961. Mrs. F. A. Fear, Littlers Farm, Wrington Hill, Wrington, Bristol. SOMERSET.
December 18th, 1961. R. Whistance, South Bank, Much Marcle, Ledbury, Herefordshire. HEREFORDSHIRE.
December 18th, 1961. L. H. Jones, Royal Content Farm, Snuff Hill, Fairfield, Bromsgrove, Worcs. WORCESTERSHIRE.
December 19th, 1961. J. W. Stennett, Place Farm, Rougham, Bury St. Edmunds, Suffolk. Disease at: Lodge Farm, Coney Weston, Bury St. Edmunds, Suffolk. WEST SUFFOLK.
December 19th, 1961. T. Isles, Willow Nook Farm, Benson Lane, Eaves, Woodplumpton, Preston, Lancs. LANCASHIRE.
December 19th, 1961. J. H. Yeoman, Glanbaiden Farm, Govilon, Abergavenny, Mon. Disease at: Werngochen Farm Piggery, Llantilio Pertholey, Abergavenny, Mon. MONMOUTHSHIRE.
December 19th, 1961. J. H. Yeoman, Glanbaiden Farm, Govilon, Abergavenny, Mon. MONMOUTHSHIRE.
December 19th, 1961. A. H. Jagger, The Nurseries, Whitehall Road, Wyke, Bradford, Yorks. CITY OF BRADFORD.
December 20th, 1961. J. R. Paddock, Whitton Farm, Westbury, Shrewsbury, Salop. SHROPSHIRE.
December 20th, 1961. D. O. Hughes, Ty Ucha, Abergele, Denbighshire.
December 20th, 1961. E. L. Walgate, Paddock House, Burton Pidsea, Hull, Yorks. YORK, EAST RIDING.
December 20th, 1961. T. G. Snowdon, 3, Council Houses, Weeley Heath, Clacton-on-Sea, Essex. ESSEX.
December 20th, 1961. S. H. Tompkins, Manor Farm, Thornborough, Buckingham. BUCKINGHAMSHIRE.
December 21st, 1961. F. J. Brooke, Great Moor Court, Much Marcle, Ledbury, Hereford. HEREFORDSHIRE.
December 21st, 1961. C. T. Fothergill, Rose Villa Farm, Melmerby, Ripon, Yorks. YORK, NORTH RIDING.
December 21st, 1961. H. J. B. Dawson, Howgrave House, Sutton Howgrave, Bedale, Yorks. YORK, NORTH RIDING.
December 21st, 1961. T. R. Clarke, 42, Church Lane, Barnham, Bognor Regis, Sussex.
December 21st, 1961. S. D. Kynaston, Broniarth, Guilsfield, Welshpool, Montgomeryshire. MONTGOMERYSHIRE.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Laboratory Diagnostic Services

Sir,—The publication of the Symposium on the Provision of Laboratory Diagnostic Services (*Vet. Rec.*, November 18th, 1961) will undoubtedly provoke a lot of discussion amongst those who use the laboratories and those who staff them. Several speakers stressed the need for more laboratories. It was suggested that the maximum working distance of a laboratory was about 50 miles. Loosmore states that two-thirds of all the farm samples received by the Reading Investigation Centre come from practices within a radius of 30 miles. I suggest that the majority of carcases submitted to laboratories probably come from inside a radius of 20 miles.

The importance of getting carcases to the laboratory in a fresh state cannot be over-emphasised. In these days of intensive stock-keeping and widespread use of antibiotics, the practitioner frequently meets outbreaks of disease which quickly build up to a high morbidity and high mortality. He cannot be sure that the antibiotic or sulphonamide he used in a similar outbreak the previous week will be effective. Most would agree that in cases of bacterial disease it is reassuring to have an early laboratory report on the drug sensitivity of the bacteria implicated. Unfortunately, the practical difficulty of transporting carcases to the laboratory frequently means that no laboratory examination is made.

One alternative is for the veterinary surgeon to send by post selected organs removed at a *post-mortem* examination made on the farm. There are important objections to this. It means the report is delayed 24 hours and the final report will not be so helpful because, firstly, it is based on the examination of a few organs extracted with non-sterile instruments, and, secondly, overnight confinement in an air-tight parcel so hastens decomposition that it often arrives in a condition quite unsuitable for examination. At this laboratory when bacterial disease is suspected in a pig or calf, a range of organs are cultured in addition to three different levels of the intestine. Sensitivity testing is carried out on any bacteria that have invaded the organs or on those multiplying in that part of the intestine worst affected by enteritis. Thus a preliminary and usually reliable report can be given on the sensitivity range the next day.

Some farmers are unable, or unwilling, to bring in dead animals. Some practitioners consider it worth while to bring in carcases themselves from distances up to 15 and sometimes 20 miles.

I wish to suggest one possible solution to the distance problem. Many practices find it convenient to run a spare car. If the spare vehicle were a light van, so cutting costs by saving the purchase tax, it could be used to deliver carcases and other specimens to the laboratory. A layman could make these journeys.

This would involve much less cost than equipping and manning a small laboratory, as has been done in a few practices in recent years. Such small laboratories, although useful, can never hope to have the resources of a fully-fledged diagnostic laboratory.

Yours faithfully,
C. T. McCREA.

Ministry of Agriculture, Fisheries and Food,
Veterinary Investigation Centre,
West House,
Thirsk,
Yorkshire.

December 14th, 1961.

The Behaviour of Cattle in Crushes

Sir,—Recent correspondence in THE VETERINARY RECORD by Ewbank and others on the behaviour of cattle in crushes prompts me to describe a behaviour pattern shown by cattle when confined in a long crush or race, which can be most troublesome and time-consuming.

On this station the cattle are ranched; and twice a week, for blood collection, rectal examination, etc., necessitated by our research programme, the cattle are passed into a 30-yard-long crush. Continually we are held up in our work by the animal that charges down the crush, adopts a kneeling submissive state and thrusts its head far forward between the legs of the animal in front.

It is always the same animals that behave in this way and I have a suspicion that it runs in families. Nothing, I find, short of emptying the crush again, will get the animal back on its feet.

Fraser (1960) describes very fully this kneeling submissive state and classifies it as a transient form of "tonic immobility." Unfortunately he offers no suggestion as to either its cure or prevention.

Yours faithfully,
J. S. MACFARLANE.

Division of Veterinary Services,
Livestock Research Station,
P.O. West Kilimanjaro,
Moshi,
Tanganyika.

Reference

FRASER, A. F. (1960). *Canad. J. Comp. Med.* **24**. 330.

Neonatal Scouring in Piglets

Sir,—I was interested to read the recent correspondence on the efficacy of neomycin against neonatal scours in young piglets.

I can confirm the results of Steele-Bodger *et al.* (*Vet. Rec.*, December 9th, 1961) as I have obtained dramatic results using neomycin sulphate at a dosage rate of 100 mg. per piglet administered by both oral and parenteral routes. I was induced to use the intramuscular form of therapy, as frequent *post-mortem* examination of young piglets showed a general septicaemia associated with the scour, and I understand that the absorption of neomycin from the intestines is poor.

I would also like to record the use of neomycin therapy in calf scours associated with *Escherichia coli* infection. I am convinced that the results obtained are equal to those of any existing form of treatment, and in many cases a higher percentage of cures has been obtained than would have been the case using other treatments.

Neomycin has also been used by me in the treatment of salmonella infections of calves and dairy cows. In one outbreak *Salmonella typhimurium* was isolated from the cows and of 4 cases treated all recovered. I used 3 grammes neomycin sulphate by intramuscular injection once daily supplemented by 2 grammes orally twice daily for 4 days. Unfortunately, however, one cow succumbed to Johne's disease a month later. Positive pre-treatment swabs were obtained from all clinically affected animals.

The calves had a *Salmonella dublin* infection, and of 15 affected, 2 were slaughtered as advanced cases before treatment commenced. Of the remainder, 12 were cured and 1 died within 48 hours of showing per-acute symptoms. Treatment consisted of 2 grammes neomycin sulphate in aqueous solution by intravenous injection on the first day followed by 1 gramme intramuscularly for the next 3 days. Oral medication with 1 gramme twice daily was used for the same period.

Positive pre-treatment swabs were obtained from only 7 calves, but the remainder showed typical and similar symptoms and were assumed to be infected. Such a picture is not uncommon in these outbreaks.

In both cows and calves clinical infection was easily recognised as also was clinical cure, but bacteriological cure was only taken to be established when weekly rectal swabs were negative on culture on 3 consecutive occasions. This criterion is open to criticism as it is well known that excretion of salmonella organisms may be intermittent, and one is conscious that a non-excreting carrier state may exist. One clinically cured calf with a positive swab had to be given further treatment and this was apparently successful.

I am indebted to the Veterinary Research Division, Boots Pure Drug Co., for supplying the neomycin, and suggesting its parenteral use, and also for doing the large amount of bacteriological work which was a necessary adjunct to the investigations.

Yours faithfully,
D. L. HAXBY.

Candant House,
Upton,
Nr. Southwell,
Notts.

December 15th, 1961.